Principles of
SURGICAL PRACTICE

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DEDICATION

To all who devote time and effort

to the teaching of Surgery

PRINCIPLES OF SURGICAL PRACTICE

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Preface

Surgery like all of medicine is advancing at a more rapid than at any previous period in the long history of man s struggle against disease. New clinical syndromes, refinements in diagnostic methods which reflect the latest developments in the physical and biologic sciences, and therapeutic measures which extend the promise of recovery to numerous abnormalities that only recently were entirely hopeless or at best, amenable to the mere palliation of symptoms now follow one another at so rapid a pace that even specialists in restricted fields have difficulty in assimilating them These advances create new social and economic problems by prolonging the life span of the earth s inhabitants and by accelerating the rapid increase in world population They further compel changes in the form of medical practice and, as a corollary in medical education

The increasing complexity of medicine in all of its divisions makes it more and more difficult for the general practitioner to become and remain proficient in all branches and it tends to hasten the trend toward specialization into ever restricted disciplines Coupled with this is the growing pressure from specialty boards upon hospitals to limit access to operating and delivery rooms to physicians whose certification attests an adequate postgraduate training and demonstrated skills Much of medicine is still practiced by the general practitioner whose indispensability is generally recognized, but his scope nonetheless, is being narrowed Medical curricula must be shaped to meet these changes, and textbooks, too must be adapted to serve them. Only by intelligent awareness of the alterations in the form of medical practice can medical education continue to discharge its heavy responsibility

In its wider definition, which embraces all of its subspecialties surgery is not merely a form of treatment. It is a large and important division of medicine complete with its diagnostic methods its specific approaches its indications and contraindications, as well as pre- and postoperative management, the operation itself and a wide range of non-operative therapeutic methods. Indeed the most competent internist must avail himself of the particular knowledge and specialized technics of examination of the various divisions of surgery in the diagnosis of complicated medical problems

Very importantly the surgeon has taken his problems into the laboratory where his investigations of anatomy physiology and pathology have not only served his particular needs, but have enormously enriched the medical sciences. This has been the tradition of surgery since its beginning While medicine was lost in the clouds of philosophy and speculation surgery was compelled to seek out the details of body structure so essential in its everyday practice It is no wonder then, that the early anatomists were surgeons, including Erasistratus and Herophilus of the Alexandrian School the great Galen of the Roman period the medieval and Renaissance figures, such as Vesalius, Eustachius, and Fallopius and the later British surgeon-anatomists such as the Hunters, the Monros, and the Bells The study of comparative and morbid anatomy and of function naturally followed and comparably significant contributions were made In recent decades experimental surgery has reached into regions and conditions that were traditionally the stronghold of the internist that is, the heart, the lungs the liver disturbances of metabolism and circulation, and now the field of mental derangement. It is with complete justice that Sir

Clifford Albutt, himself a distinguished physician as well as a historian has called surgery 'the scientific arm of medicine

The question as to how much of surgery and what phases of it should be taught to undergraduate medical students is a timely and pressing one In full recognition of the facts that only a small fraction of any medical class will ultimately become surgeons and that these few will have to attain their knowledge and skill during the course of their long periods of specialized training it is still our conviction that some introduction to surgery should be part of the general education of all medical candidates As internes, at least one half of their duties will be in the care of surgical patients Furthermore for those whose ultimate aim is general practice or specialization in other fields some knowledge of the surgical problems and approaches is an essential part of their preparation.

It is obvious that the undergraduate exposure cannot make the student a surgeon Matters of technic therefore, should be of no value to him What is important is that he have some awareness of the principles underlying surgery and these principles are in essence a restatement of the basic sciences, anatomy physiology microbiology chemistry and pathology as they are applied in understanding and treating the diseases which come within the purview of the surgeon We believe that the integration between the basic sciences and the clinical subjects so universally sought by current medical educators could most naturally be brought about if the preclinical instructors from the first day in medical school, could project constantly the application of facts being taught to ultimate patient care and if the clinicians, in turn would continuously refer back to material covered in the preclinical years.

The second general concern of medical educators is the overcrowding of the curriculum which is intensified by pressures to reduce the time now required for the preparation of the physician for the practice of medicine. Here it would appear the elimination of unessential material and the attempt to substitute *education* for technical training would help to accomplish this purpose This could be accomplished by a thorough grounding in the underlying principles, in place of the memorizing of an endless series of more or less unrelated facts.

As a corollary to this bedside teaching should be increased and didactic programs correspondingly restricted. A general introductory course seems necessary to serve as framework upon which the facts later to be learned in the wards and laboratories are to be appended For this purpose, a textbook which supplements the classroom instruction is needed. This book was prepared to serve this purpose

There are numerous surgical texts available These are of such excellence that an attempt to improve upon them would seem to be presumptuous. However in the light of what has been said above, these books do not seem to be relevant to the needs of the undergraduate medical student They are in the main voluminous and encyclopedic treatises far beyond the scope and the capacities of the students to assimilate in the time allotted to the subject The very bulk of their material is a deterrent to their reading If the student had nothing else to do during the months devoted to his introduction to surgery he would be unable to encompass them And when, at the same time he has even larger books on medicine and equally overwhelming texts on obstetrics, pediatrics and other clinical subjects, the total reaches the point of becoming ludicrous.

We believe that a text which would complement the extent of the material covered in the hours spent in the classroom should be available This does not mean a compendium but rather a book devoted solely to the *principles* mentioned above The undergraduate should be helped to understand the disturbances involved and their correction in terms of alterations in the underlying anatomy and physiology rather than to memorize syndromes and therapeutic methods Above all together with the atti

to trust his thinking rather than his memory processes. Finally, the critique conducted after the examinations have been read and criticized constructively by the instructor attempts to insure that this, indeed has been the case To help attain these aims a book should be readable clear and short enough to be covered together with classroom instruction. To the extent that these requirements succeeded in their purpose

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1

Inflammation and Wound Healing

I *Inflammation*
  A Definition
  B Characteristics (1) the vascular phenomenon and cellular elements (2) the inflammatory exudate
  C End results of the inflammatory process
  D Symptoms of inflammation
II *Wound healing*
  A Specialization of cells
  B Inhibiting factors (1) local (2) systemic
  C Types (1) first intention (2) second intention (3) secondary closure (4) grafting

INFLAMMATION

Definition

Inflammation is the total body response local and general, to the cellular damage caused by an irritant It is a basic phenomenon in the pathogenesis of many disease processes seen by the surgeon and its varied manifestations form an integral part of surgical pathology

The *inflammatory process* embraces the series of changes by which the organism reacts to injury defends itself against invasion and restores toward normal those tissues which have been damaged by irritants of any nature The irritants which evoke this response include most of the disease-producing agents with which the body contends. Among these are the pathogenic microorganisms, trauma, thermal and actinic agents, chemical substances, and, occasionally neoplastic processes Inflammation plays a key role locally and systemically in the pathologic changes and in the clinical manifestations which are the concern of the surgeon In some forms of irritation such as those produced by trauma or chemical agents, the cellular damage is inflicted immediately after which repair begins. In bacterial inflammations, the noxious effects tend to progress unless or until they are checked by the defense mechanisms of the host.

Characteristics

The Vascular Phenomenon and Cellular Elements. When tissues are injured the defensive and reparative processes are immediately mobilized. Earliest in this sequence of events is an increased blood supply to the area involved This *active hyperemia* is mediated by the release of a tissue product known as *Lewis s H substance* (H stands for histamine like Menkin has shown that these materials are polypeptide like substances which he has called leukotaxine and a euglobulin released from the injured cells.) These agents produce a dilatation of the small vessels in and adjacent to the injured tissues and increase permeability of the capillaries in this area. The outflow of fluid from the

vascular bed (*exudation*) into the interstitial space results in *increased tissue space pressure* which is responsible for slowing of the blood flow in the small vessels in the traumatized area This slowing is conducive to intravascular thrombosis in the venules and capillaries, which may lead subsequently to cellular anoxia and necrosis Formed elements in the blood are brought to the affected tissues by a process called *chemotaxis* they help in delimiting the area into which an invader might penetrate, and they perform the function of *phagocytosis*

The Inflammatory Exudate *Inflammatory exudate* is the name given to all of the extravasated elements that have been summoned to do battle with the invader It includes three essential components (1) fluid which is derived from both blood and lymph (2) cellular elements consisting of leukocytes and macrophages (3) fibrin a protein substance derived largely from lymph. Ultimately it includes the products of tissue necrosis The leukocyte fraction is predominantly polymorphonuclear in acute bacterial infections a relative increase of eosinophils is found in the more subacute and chronic infections and in response to acute and subacute allergic irritants Lymphocytes characterize the response in chronic diseases Macrophages are derived from the fixed tissue cells (histiocytes) and from the reticuloendothelial system when there is particulate matter to engulf and carry away

End Results of the Inflammatory Process

The localization of an infection depends upon the relative virulence of the invading agent and the totality of the defensive forces which can be mustered against it. One factor is the presence in adequate amounts of substances such as fibrin and mucoproteins, which aid in walling off the process The effectiveness of the available fibrin is influenced by fibrinolytic substances which may inhibit its action The character of the invading organism is important Staphylococci which produce little fibrinolysin cause infections which are readily limited by the inflammatory process (walled off by a *pyogenic membrane*) whereas streptococcus infections characteristically become diffuse and spreading because of the specific fibrinolysins which these organisms produce *Clostridium welchii* infections spread most rapidly for a similar reason Other substances, such as hyaluronidase which are produced by the common microorganisms may also tend to prevent localization and lead to spreading infections.

When an infection has become localized, as usually occurs in those of staphylococcal origin *suppuration*—the formation of pus—takes place The necrotic and fluid material within the pyogenic membrane is called *pus* and contains serum fibrin, leukocytes, macrophages foreign material, and necrotic tissue *This material, together with the wall* that encloses it constitutes an *abscess* When the inflammatory exudate fails to limit the bacterial invasion, the infection spreads through loose connective tissue and along the planes of fasciae and muscles This type of infection is called *cellulitis* When abscesses form surgical therapy consists of incision and drainage of the purulent material In cellulitis, local intervention is usually futile and often harmful. The treatment must remain systemic and includes the giving of antibiotics which may be brought to all of the tissues by the blood stream and which will combat the bacteria at any point to which they may have spread.

Certain immunologic processes, both local and systemic, as yet incompletely understood, aid in the overcoming of infections. The facility with which incisions in certain areas heal notably those of the head and of the perineum indicates a tissue resistance and/or immunity to the patient s own bacterial flora Activity of the reticuloendothelial system and the formation of gamma globulins are involved in systemic immunologic processes.

Symptoms of Inflammation

The classic local symptoms of inflammation are pain, heat, redness, swelling and disturbed function. Pain is the result of pres-

sure of the inflammatory exudate upon nerve endings and of irritation from tissue and bacterial metabolites and from foreign bodies. Heat is the result of the active hyperemia bringing an increased circulation to the area redness is due to the hyperemia and the dilatation of blood vessels Swelling is the result of the inflammatory exudate in the interstitial spaces Throbbing a pounding sensation synchronous with the heartbeat, is common

The systemic symptoms and signs reflect the mobilization of the total defenses of the body to the inflammatory agent They include fever tachycardia, tachypnea leukocytosis and occasionally chills The latter are due to the absorption into the circulating blood stream of bacteria or toxic materials

WOUND HEALING

Surgery is constantly concerned with the process of wound healing whether with traumatic wounds or with those produced during surgical intervention The incisions through the skin and through the musculofascial lamina of the abdomen the opening into the bowel preparatory to anastomosis with another segment of bowel the ligation of blood vessels, the closure of a parenchymatous organ from which a portion has been removed for pathologic examination—all ultimately depend upon the process of wound healing This is a continuing process and is not completed until anatomic integrity and physiologic function are restored

Specialization of Cells

There is a marked difference in the ability of various types of cells and tissues to reproduce themselves Epithelium including skin, does so readily Skeletal muscle is replaced by cicatrix, as are also parenchymatous organs Certain solid organs, notably the liver have a marked ability to regenerate their kind in order to compensate for loss that has been incurred but this is distinct from the process of healing. Tendon which is a nonspecialized tissue replaces itself as tendon. Cartilage replaces itself poorly if at all Bone heals itself by a complex process including *callus formation* which is discussed in detail in Chapter 7

Inhibiting Factors

Local Failure of the normal process of wound healing may indicate deficiencies which are local or systemic The local factors which tend to inhibit wound healing are the following (1) insufficient blood supply to the area the diminished blood supply may have been produced by the operative ligation of an excessive proportion of the arterial blood supply to the part however it may be due to generalized disease as well, such as arteriosclerosis (2) infection which may destroy the elements that contribute to wound healing (3) inadequate drainage of necrotic material which inhibits the normal process of wound healing by preventing the obliteration of dead space and the reapposition of wound edges (4) inadequate hemostasis which leads to separation of injured surfaces by accumulations of blood or serum (5) failure of accurate apposition of edges (6) extraneous foreign material of any kind which may perpetuate infection and so delay healing

Systemic Systemic causes of failure or delay in perfect wound healing are as follows (1) hypoproteinemia—an inadequate total circulating serum protein level (2) deficiency in circulating hemoglobin restoration of hemoglobin levels commands priority over all other functions in the utilization of administered proteins (3) wound healing is inhibited in the absence of a normal serum vitamin C level there is very little storage of vitamin C in the body it is heat labile vitamin C blood levels are found to be low in general debility in dietary deficiency and scurvy during fever and during great stress (4) agammaglobulinemia or hypogammaglobulinemia also results in failure of normal wound healing

Types of Healing

Whenever a wound is inflicted, a sequence of events is immediately initiated which ter

minates ultimately in healing This process *includes the phenomenon described above as inflammation* There is increased vascularity *exudation of plasma* and later prolifera tion of fibrous tissue

Healing by First Intention If the wound edges are approximated the process of re pair is limited to the gap between the ap-posed surfaces and tends to unite them with a minimum of scar formation The inflam-matory exudate forms a coagulum which cements together the edges of the wound If there is gaping, external aids, such as su-tures are used to hold the edges mechani cally in apposition Within 3 to 5 days the phenomenon of fibroplasia takes place within this coagulum in which new blood vessels are laid down and connective tissue is formed This occurs by the proliferation of fixed tissue cells to form capillary buds, which grow out into the coagulum Later immature fibroblasts are laid down about the newly formed blood vessels This mate rial is called *granulation tissue* Beginning about the seventh day and proceeding there after toward completion at about the four teenth day the nuclei of the fibroblasts are lost to the cells and the blood vessels begin to disappear by retraction of the fibrous tis sue The result is a cicatrix, or a scar The progress of this sequence of events without interruption delay or distortion is called *healing by first intention*

Healing by Second Intention. When the edges of a wound are not in apposition the defect heals from its base upward This may be the result of failure to suture the wound or of loss of substance from trauma or in-fection The end result is obliteration of the defect by a broad scar rather than the union of normal tissues by means of a fine cicatrix The process of granulation is the same but greater in amount as the wound heals from below upward Healing by second intention is allowed to proceed purposefully whenever it is not considered safe to coapt the edges because of the danger of infection from gross contamination or excessive lapse of time Healing by second intention allows necrotic material debris dirt, or foreign ma terial to be extruded from the wound as the wound "granulates in" from below

Secondary Closure When the surgeon is confronted with a wound that he is reluc tant to suture because of possible contam ination, delayed closure may be resorted to The wound is allowed to remain open for a period of 2 3 or 4 days, at the end of which interval if it appears bacteriologically clean, it may then be secondarily sutured. Closing the wound at a late stage after it has begun to heal by second intention is called *healing by third intention*

Grafting A fourth method of wound heal ing is applicable to surfaces which have been so extensively denuded that under no con ditions can the edges be apposed Healing by second intention would not only result in a large and deforming scar but a long period of time would elapse before the wound would be closed Under such conditions skin grafting is a valuable adjunct. A piece of skin either partial or full thickness, may be taken from another part of the body and placed upon the wound, thus closing it per primam with a graft. Grafting may also be done in later stages to close extensive gran ulating surfaces Tissues other than skin may also be grafted and in reconstructive sur gery frequent use is made of the technics of grafting skin muscle tendons nerves blood vessels and bone

SUMMARY OF PRINCIPLES

1 Inflammation is a process characteristic of biologic tissue which per mits it to defend itself against an invader and which initiates the process of repair as a step toward restoration to health

2 Characteristic of the process of inflammation is the inflammatory exudate which consists of fluid cells and fibrin In and by virtue of this

milieu inflamed or destroyed tissue is replaced by like tissue or by scar

3 Basic local and generalized factors from the setting up of local mechanical barriers to systemic immunologic and antibiotic processes determine the limitations of the inflammatory process

4 Local factors that inhibit wound healing are an insufficient blood supply inadequate drainage the presence of necrotic tissue or foreign body, infection inadequate hemostasis and poor apposition of edges

5 Systemic factors that inhibit wound healing include hypoproteinemia hypohemoglobinemia low blood vitamin C level agammaglobulinemia, or hypogammaglobulinemia

6 Wound healing proceeds along one of several lines healing per primam (by first intention) healing by second intention healing by third intention or healing by grafting of large wounds

SUGGESTED READING

WILLIAMSON M B ed. *Healing of Wounds* McGraw Hill Book Company Inc. Blakiston Division, New York, 1957

2

Surgical Microbiology and the Management of Infection

I. Introduction: the meaning of the above title
II. Pathogenesis: sources of contamination and spread within the body
III Operating room technic
IV The mechanisms of postoperative infections
   A The wound itself
   B Respiratory tract
   C Urinary tract
V Surgical biotherapy and chemotherapy
   A General statement
   B Serous cavities (1) pleurae (2) peritoneum (3) pericardium (4) meninges (5) synovia (6) tendon sheaths
   C Soft tissue infections (1) cellulitis (2) abscess (*a*) furuncle (*b*) carbuncle (3) infections of the hand (*a*) Paronychia (*b*) felon (*c*) tendon sheaths and fascial spaces (4) gas gangrene and tetanus
   D Infections of bone
   E Specific pathogens and the diseases they produce (1) bacteria (2) fungi (3) viruses

INTRODUCTION: THE MEANING OF THE ABOVE TITLE

The transition from microbiology as a separate discipline to its application in clinical surgery is difficult for the student. Surgical microbiology concerns itself with the disease forms produced by pathogenic microorganisms and the methods for their control. In addition it must deal with the surgical technics for operating without introducing infection into previously uncontaminated tissues.

The fields of clinical surgery microbiology and pathology are intimately interwoven with each other The rapid and continual changes in the field of microbiology must be closely followed in their application to human disease Medical science and particularly the development of biotherapeutic and chemotherapeutic agents, has greatly modified the natural history of infectious disease and the characteristics of infective organisms. Even new diseases and problems in clinical surgery are being created and uncovered by developments in the field of clinical microbiology

PATHOGENESIS. SOURCES OF CONTAMINATION AND SPREAD WITHIN THE BODY

In the never-ending warfare between man and the microscopic world the defenses of

the body may be reached through various portals. These include the skin, nasopharynx and alimentary and urogenital orifices. Contamination may reach an accidental or surgical wound by contact with foreign surfaces, air borne particles, or the hands and nasopharyngeal secretions of the surgeon and his team. The transition from contamination (the entrance of pathogens) to infection (the morbid changes produced by their survival and proliferation) is dependent upon many factors. These include the element of *time*, the *severity* and *duration* of the *period of exposure*, and the relation between *virulence* of the organism and *resistance* on the part of the host. It is well known that a single contamination, for example, of the peritoneal cavity by intraluminal intestinal contents may be very well handled by its remarkably defensive mesothelial lining; this is likewise true of the skin. However, repeated contamination of the peritoneal surface or of the n over a long period of time is a much re serious matter in that spread and owth of microorganisms within the human st is permitted. The time element is of rticular significance in accidental wounds. the wound is treated properly after its infliction, the initial contamination is over come and infection prevented. If, however, a number of hours elapse (the "golden period") the contaminating organisms have had the opportunity to penetrate and proliferate and give rise to wound infection. Eventually, if enough time has elapsed, remote dissemination of the infection through the lymph and blood streams may occur and compound the problems of therapy.

OPERATING ROOM TECHNIC

Throughout the long history of surgery the ever present hazard of infection constituted one of the principal barriers to the development of operative therapy. Hygiene, as we understand it, was nonexistent. Semmelweis demonstrated that childbed fever was carried to the parturient woman by the contaminated hands of the doctors and that this dread disease could be prevented if those in attendance merely washed their hands. Even this knowledge of the relation of dirt to infection was not applied to surgery. However, when Pasteur proved that disease was caused by microscopic organisms, the way to the control of infection was opened. Lister showed that preventing the access of bacteria to the operative wound protected the patient from infection and death, and he also became immortalized as a benefactor to mankind. In his initial efforts he attempted to kill the germs with disinfectants before they could reach the wound. The method was cumbersome and unpleasant in that both surgeons and the patient breathed a fog of carbolic acid spray. (Many skin antiseptics are now available which destroy normal tissue minimally but have a high coefficient of destruction or inhibition of microorganisms.) Thus was initiated the era of antiseptic surgery, which soon gave way to the aseptic technic which we use today. This is based on the concept of preventing the access of bacteria to the wound. The introduction of sterile rubber gloves and the wearing of masks by Halsted was a further refinement. By the use of aseptic technic, including sterilization by the improved process of autoclaving, the feats of modern surgery have been made possible.

THE MECHANISMS OF POSTOPERATIVE INFECTIONS

The Wound Itself

Postoperative infection of an accidental or surgical wound may be due to either local or systemic factors. Local failure may consist of an initial grossly contaminated accidental wound, the introduction of contamination in a surgical wound, a lapse of time beyond the "golden period" between contamination and treatment, a failure to remove foreign bodies and necrotic debris, and a break in aseptic technic. (Proper wound

treatment will be reviewed in Chapter 3) Systemic causes of failure include all the factors which tend to inhibit normal wound healing plus inadequate immunologic response

Respiratory Tract

There are two additional body systems which are commonly the site of postoperative infections the respiratory system and the urinary system Postoperative infections of the respiratory system are not only frequent and serious but until recently pneumonitis constituted one of the principle causes of death The predisposing factors are instrumentation of the respiratory tract, aspiration of vomitus and disturbed physiology of the respiratory system. Instrumentation includes endotracheal intubation for anesthetic purposes and tracheobronchial suction for the removal of excessive secretions. The danger of aspiration of gastric contents into the bronchial system is always present when the normal defensive reflexes are obtunded by anesthesia. This makes for the importance of preoperative fasting, the use of nasogastric suction in gastrointestinal obstructions, meticulous attention to reflex vomiting and swallowing during anesthesia and the use of the parenteral route for alimentation during the early postoperative period.

The most important predisposing factor in postoperative infection of the respiratory tract is the disturbance of physiology of respiration following operations in the thorax or upper abdomen. Respiratory excursion is limited because of pain and the voluntary splinting of the thorax on the ipsilateral side Because of the shallow breathing the lower reaches of the lung may then become atelectatic, since the inspiratory effort is not vigorous enough to distend the alveoli Two other predisposing factors in the development of atelectasis are elevation of the diaphragm consequent to abdominal distention and excessive sedation of the patient in the early postoperative period Both factors act by leading to shallow respiratory excursion and the inadequate alveolar distention which follows Atelectasis is a common precursor of pneumonitis

The treatment of the postoperative respiratory infections begins prophylactically with adequate preoperative preparation of the patient This should include the proper examination of the lungs and the treatment of any pre existing pulmonary condition such as retention of secretion especially if suppurative which might predispose to pulmonary edema and to pneumonitis The active treatment of the postoperative pulmonary infection is the resumption of function of the atelectatic lung This is most efficiently accomplished by eliminating pain which is the cause of the shallow breathing Thoracic and high abdominal pain are effectively relieved by local anesthetic block of the appropriate intercostal nerves With restoration of normal respiratory excursion, atelectasis, retention of secretions, and pneumonitis are less common. Tracheobronchial toilet may be accomplished by the aerosol administration of bronchodilators and wetting agents which will remove the accumulated material in the smaller bronchi and alveolar spaces. Bronchoscopy and direct aspiration of the bronchi under vision may occasionally be resorted to

Urinary Tract

Urinary tract infections are also frequent in the postoperative period Predisposing factors are stasis or urinary retention due to reflex disturbances in the neuromuscular control of micturition and the introduction of contaminants by repeated or prolonged catheterization Certain drugs, notably morphine may contribute to the difficulty in emptying the bladder Poor perineal hygiene in the female in the postoperative period is an additional cause of ascending urinary tract infection Treatment of postoperative urinary tract infections prophylactically includes the

correction of stasis by early ambulation and the avoidance of unnecessary catheterization postoperatively it includes the avoidance of drugs which cause overdistention mediated through spasm of the sphincter at the outlet of the bladder In addition diuresis and the employment of urinary tract antiseptics and antibiotics are helpful Such agents must be selected according to bacteriologic examination of the urine including identification and sensitivities of the involved organisms.

SURGICAL BIOTHERAPY AND CHEMOTHERAPY

General Statement

The chemotherapeutic and antibiotic agents have assumed a dominant role in the management of surgical infections, and the need for operative intervention has been correspondingly reduced Major infections require attention to the general body economy which implies the maintenance of optimal physiologic norms, including nutrition fluid-electrolyte balance and blood profile Antibiotic and chemotherapeutic agents are selected on the basis of their effectiveness against the invading organisms as determined by culture and sensitivity tests. Initially while awaiting laboratory guides to therapy empiric selection of antibiotic agents (or presumptive selection on the basis of high probability index) is necessary Active surgical intervention is employed mainly for the purpose of removing necrotic material and debris (pus) by providing access to the outside of the body

Serous Cavities

The treatment of infections of the serous cavities—pleural peritoneal, pericardial synovial—follows certain principles which are common to all, plus others that vary with the particular cavity affected In general the broad absorptive surface makes for severe systemic disturbances All are closed spaces, which necessitates providing for drainage of purulent material Following are brief descriptions of the methods in which clinical biotherapy and chemotherapy are employed in the treatment of serous cavities Each will be discussed in greater detail in subsequent chapters

Pleural empyema is a suppurative infection of the potential pleural space The most common pathogenetic agents are (1) pneumonia (not nearly as common as before the antibiotic age) (2) accidental wounds of the chest and (3) surgical intervention into the chest Three factors peculiar to pleural cavity infection must be recognized for their influence in directing the control of such processes. First is the question of the ability of antibiotics and chemotherapeutic agents to traverse the serous lining of the cavity Second the presence of a fistula between a bronchus and the pleural space may constitute a source of perpetuation of the contamination Finally there is the problem of a rigid chest cage which obviates the ability of the wall to approximate itself to the viscera and thus to obliterate a cavity Parenthetically failure of obliteration of an abscess cavity wherever it may occur is an important hindrance to healing of the infection Empyema can frequently be treated by nonoperative means, i.e., with the withdrawal of fluid by needle and syringe and the administration of antibiotics both parenterally and intrapleurally Further spread and the sequelae of the infection may thus be avoided If the empyema cannot be definitively cured by these methods, surgical evacuation of this pleural abscess must be undertaken. If a pyogenic membrane has already been formed on the visceral surface on the pleura encasing the lung (fibrothorax) and is preventing its re-expansion it may be removed by the procedure known as *decortication* Whereas the handling of so much infected material in the preantibiotic day would almost certainly have led to failure and

death success and recovery from these infections is the rule today Enzymatic debridement, the introduction of certain fibrinolysins intrapleurally discussed at greater length in Chapters 16 and 17, may be used prior to decortication and may bring about resolution without operation Obliteration of cavities by operative technics which collapse all or part of the chest wall (thoracoplasties) is sometimes necessary

Peritonitis is an infection of the lining membrane of the abdominal cavity Biotherapy and chemotherapy in this disease are only adjunctive to the treatment of the underlying causative disease Antibiotics play an important role in the prophylactic management of peritonitis, whether they are used preoperatively to sterilize the intestinal tract or administered before an infective process has reached the peritoneum. Preoperatively the nonsoluble sulfonamide drugs are particularly effective against the *Escherichia coli* group the symbiotic hemolytic streptococcus responds better to streptomycin Neomycin is most effective in the shortest period of time in sterilizing the intestinal tract against virulent pathogens in the preoperative period Preoperative bowel antisepsis inhibits the formation of vitamin K by the normal intestinal flora adequate amounts of the vitamin must therefore be administered simultaneously

The definitive therapy of peritonitis is directed primarily against the disease process which has caused the peritonitis such as a ruptured solid viscus spilling blood or bile into the peritoneal cavity or perforation of a hollow viscus Also necessary is the treatment of the toxemia and the anemia consequent upon such massive infection. The disturbances of fluid and electrolyte balance are countered vigorously as is detailed in Chapter 5 Until sensitivity determinations are available the most effective parenteral antibiotic therapy continues to be the combination of sulfonamides against the coli form group and streptomycin to combat the microaerophilic streptococci

Pericarditis is an infection in the space between the visceral (epicardium) and the parietal pericardium Acute suppurative pericarditis is rare in the antibiotic age It may be metastatic from distant infection or may occur by extension It may also follow operation or trauma. In addition to the danger from infection which is combatted by systemic antibiotic therapy there is the added serious hazard of cardiac tamponade—the excessive pressure of a large exudate embarrassing cardiac dynamics Therefore, aspiration of the pericardial sac fluid both for diagnosis and for therapeutic relief of tamponade is frequently performed A late sequel to acute pericarditis may be the formation of adhesions between the visceral and parietal pericardial layers which may result in a fibrous encasement of the heart, chronic constrictive pericarditis. Chronic pericarditis is usually tuberculous in origin.

Suppurative meningitis may be blood borne (epidemic or metastatic forms) It may also result by extension from external wounds or from suppurative processes within the brain or in adjacent structures, such as the mastoid or nasopharyngeal facial sinuses The meninges, contrary to the other serous membranes, are not permeable to penicillin and only relatively permeable to other antibiotics. Sulfonamides, however readily penetrate this barrier

Suppurative arthritis is far less common than it was in the preantibiotic age It is usually secondary to infections of soft tissue wounds, which today are avoided by the wide and early use of antibiotics Acute suppurative arthritis more commonly involves the weight bearing joints, i e knee hip and ankle but is also found in the wrist, elbow and shoulder Treatment is directed toward control of the infection and preservation of function Specific antibiotic therapy is aided by aspiration, identification, and sensitivity studies of the infecting organism The principles of management of the joint infection include splinting to supplement nature s attempts to immobilize the limb surgical drain-

age and intraarticular injection of antibiotics, and the breaking up of adhesions (synechiae) within the joint

Tendon sheaths are serous-lined cavities which provide free motion for the enclosed tendon Preservation of this motion is essential to function of the digit. Pressure within the sheath from the purulent exudate may so compress the blood supply as to cause necrosis of the tendon This plus the direct destructive action of the infection leads to adhesions within the sheath which rob the tendon of its function Accordingly infection of tendon sheaths must be submitted to incision and drainage as soon as the diagnosis is made with no delay of therapy until systemic antibiotics can combat the infection. Infections of the flexor tendon sheaths of the hand are of exceptional significance because of their relative frequency and the severe disability which they cause They will be discussed later in this chapter

Soft Tissue Infections

Cellulitis is a diffuse spreading infection within the tissues The causative organism is usually the streptococcus Accompanying it there may be red streaks that course along the skin from the original site of infection to the lymph nodes which drain the area, constituting a *lymphangitis* the involved swollen, and tender lymph nodes constitute a *lymphadenitis* Operative therapy is not only useless but contraindicated treatment is by systemic administration of the antibiotics, preferably sulfadiazine intravenously streptomycin intramuscularly or one of the broad spectrum antibiotics. General measures, including immobilization and supportive care as outlined for all serious infections, are indicated

An *abscess* is an accumulation of suppurative material surrounded by a pyogenic membrane It is usually staphylococcal in origin, although other pyogenic bacteria *may* be causative An abscess represents an infection in which the defensive capacities of the host exceed the invasive powers of the microorganism The liquid contents are composed of necrotic tissue debris, living and dead leukocytes and organisms macrophages and their engulfed foreign material, and tissue fluid The pyogenic membrane which delimits the infection is an infiltration of leukocytes chiefly polymorphonuclear neutrophils surrounded by plasma cells and lymphocytes, all enmeshed in a rich fibrin network

There is an infinite variety of abscesses, depending on site degree causative organism, and symbiotic infection. However certain general principles govern their treatment. The necrosis of tissue within the abscess prevents systemically administered antibiotics from reaching the depth of the cavity Therefore management consists in aiding the localization of the infection by the use of rest, locally applied heat, and antibiotics. When necrosis and liquefaction are demonstrable by a feeling of softness in the center of the indurated swelling (fluctuation) localization has occurred and the abscess should be incised and drained Evacuation of the products of suppuration permits obliteration of the cavity by granulation and cicatricial healing General measures include supportive therapy as for any extensive infection biotherapy to prevent dissemination of the infection into adjacent tissues and into the blood stream and attention to associated diseases

A *furuncle* or *boil* is a specific type of abscess it is a suppurative infection of a cutaneous hair follicle The *Staphylococcus aureus* is the most frequent pathogenetic agent The presence of more than one furuncle or the repeated occurrence of furuncles in an individual, is termed *furunculosis* Patients peculiarly susceptible to *S aureus* infections may occasionally be desensitized with staphylococcus toxoid The antibiotic of choice is penicillin

A *carbuncle* is an interesting infection in that it represents both a partially localized suppurative process at the periphery of

which there is a spreading cellulitis of the skin and subcutaneous tissues The infection begins in a hair follicle, and it is caused by *S aureus* Carbuncles are most common in diabetics, in whom sensitivity to *S aureus* infection is generally increased In contrast to a furuncle the carbuncular infection is not successfully localized or walled off by the tissues of the host Therefore it partakes of the characteristics of both abscess and cellulitis Extension of the suppuration to the subcutaneous tissues allows it to spread centrifugally Tissue necrosis and pus formation accompany this spread The suppuration extends to the surface through adjacent hair follicles, giving the appearance of multiple confluent furuncles, draining to the surface as through a sieve

Since a carbuncle is not completely localized, it is futile to wait for fluctuation as an indication for surgical incision Concurrently with suppuration in the older area there is extension of the infection to new tissues Also since the pus-containing cavity has many recesses, simple incision will not tap the well of pus Accordingly in addition to cruciate incisions perpendicular to the surface flaps of skin and subcutaneous tissues, extending to the periphery of the indurated area, must be elevated by incisions parallel with the skin surface General care includes accurate management of the associated diabetes, if present.

Infections of the Hand

The hand demands and deserves special attention In primates its prehensile nature permits of many useful functions and is necessary for survival in nature The intricate movements performed by the fingers and hands of human beings are of the greatest importance in the economic existence of the individual and in his many esthetic accomplishments These are made possible by the beautifully complicated interweaving of muscles, tendons, pulleys nerves, and bursae Since the hands are in almost constant activity during the process of earning a livelihood and in the enjoyment of life they ar quently exposed to injury and to infe Meticulous attention to details and adhe to principles can often make the diffe between restitution or loss of delicate tion, which may be marginal in the pat economic or professional life

An acute *paronychia* (run around) suppurative infection surrounded by an of cellulitis along the proximal and l borders of the fingernail and extendin tween the bed of the nail and the soft t of the dorsum of the finger Treatment is employed early rather than late in ord avoid excessive destruction of tissue effected by the elevation of a flap of t on the dorsum of the finger separating flap from the underlying nail bed, thus leasing the pus A chronic paronychia result from an inadequately treated suba paronychia it may also be a nonsuppur granulomatous lesion, the result of a fu infection

A *felon* is the suppurative infection of volar tuft of the distal phalanx of a dig a closed space Just as in the case of t synovitis, the local treatment of a felon n not be delayed too long while parenteral tibiotic therapy is being employed The son is much the same the anterior clo

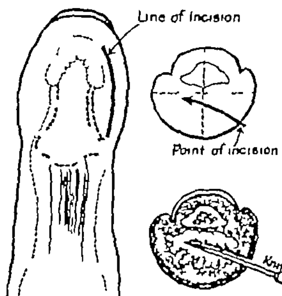


FIG. 1 Incision for drainage of felon (inf tion in volar closed space of terminal phalan (*S Koch J A M A* 4 6 1929)

space of the distal phalanx is traversed by fibrous septa extending from the bone to the skin The blood vessels entering this space not only supply the soft tissues but also include the nutrient artery to the distal two-thirds of the distal bony phalanx Incision and drainage (Fig 1) must be accomplished early in order to avoid necrosis of tissue due to obliteration of incoming blood supply by excessive interstitial pressure Incisions are made at the sides of the distal phalanx somewhat more dorsal than volar in order to avoid nerves and vessels and in order to avoid an incision across the very tip of the finger lest the tactile sensation be destroyed

The tendon sheaths of the fingers of the hand are most often involved by infection Common causes are (1) traumatic implantation of infecting microorganisms by a penetrating object and (2) direct extension of an infection from adjacent soft tissue The flexor tendon sheaths extend from the distal palmar crease to just beyond the distal interphalangeal joints. The proximal end of the flexor tendon sheath of the index finger overlies the thenar fascial space of the palm the proximal ends of the flexor tendon sheaths of the third and fourth fingers overlie the mid palmar fascial space of the palm The tendon sheaths of the flexors of the thumb and of the fifth finger are elongated and extend onto the wrist beneath the anterior annular ligament (radial and ulnar bursae) they almost always communicate with one another at the wrist. These surgical anatomic factors are important in that they contribute to the principles of treatment of infections of the tendon sheaths of the hand and of the fascial spaces of the palm

When a flexor tendon sheath is infected, either by direct implantation or by extension from soft tissue distention of the sheath with inflammatory exudate and later with pus causes acute tenderness owing to the increased pressure within a closed space The finger is painful, particularly over the course of the flexor tendon, and the patient resists even gentle flexion and extension of the finger by the examiner a bulge can frequently be seen to outline the sheath. All other associated signs of infection are present Not only may the tendon sheath and its enclosed tendon be destroyed if early incision and drainage of the pus under pressure is not accomplished, but another complication may ensue This relates to the fact that the proximal ends of the tendon sheaths overlie the fascial spaces of the hand (Fig. 2) In the case of the flexor tendon sheath of the index finger its proximal end may rupture into the thenar space which is found deep in the palm beneath the flexor tendons This catastrophe is heralded by an increased swelling and prominence of the radial side of the palm with an obliteration of its normal concavity In the case of infections of the flexor tendon sheaths of the third and fourth fingers spread from the tendon sheaths into the midpalmar space is accompanied by swelling of the ulnar two-thirds of the palm with a convexity replacing its normal concavity Infections of the long flexor of the thumb and of the flexors of the fifth finger will extend into the radial and ulnar bursae, respectively and then proceed under the volar annular ligament to the ulnar bursa compartments in the wrist. Pain and stiffness will be present in the wrist and evidence of cellulitis in the forearm.

In drainage of the flexor tendon sheaths of the fingers and the fascial spaces of the palm there are certain sites for incision that are preferable (Fig 2) Their location is designed to accomplish the desired purpose emptying of the tendon sheath of its contained pus, with a maximum of safety for the vital vascular and nervous structures of the hand. The tendon sheaths of the third fourth, and fifth fingers are incised on the ulnar border of the digit with retraction of the digital vessels and nerves dorsally an incision placed too far dorsally will cut these structures, and an incision placed too far volarward will cross the flexion creases of the fingers and impair their function. The tendon sheath of the index finger is incised on its radial surface A soft rubber drain is

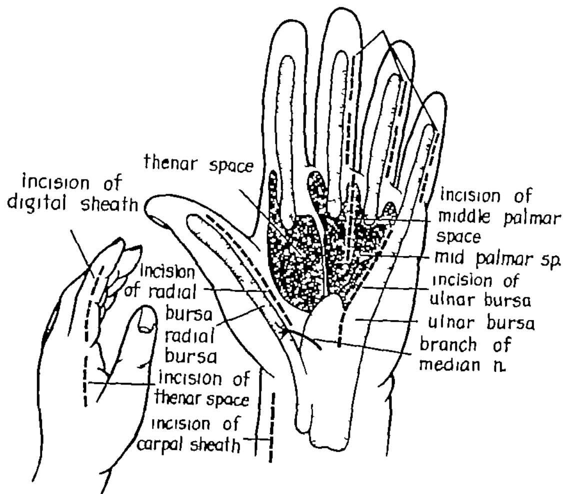


FIG 2. Topography of fascial spaces of the hand and of flexor sheaths in the hand, fingers, and wrist. Proper sites for incision of these synovial sheaths are indicated. For the index finger flexor sheath the radial side and for digits III IV and V the ulnar sides of the fingers are preferred. (*Modified from Kanavel & Mason*)

placed in the tendon sheath to insure egress of purulent material it may usually be removed within 24 hr Through-and through incisions and drains are neither necessary nor desirable An infection of the midpalmar space may be incised on the palmar surface just proximal to the web between the third and fourth digits It may also be opened through the distal palmar crease An infection of the thenar space is best drained not on the palm but on the dorsum of the web between the thumb and the second meta carpal Infection of the ulnar bursa in the wrist, an end result of an infection in either the fifth finger or the thumb is best drained by an incision on the ulnar side of the wrist extending from just above the styloid process for about 2 in. proximally The incision is carried as close to the ulnar bone as possible thereby entering the compartment which is dorsal to all the flexor tendons. A soft rubber drain is placed in this compartment and left for about 24 hr to ensure egress of purulent material Through-and-through incisions and drains are neither necessary nor desirable

Infections of soft tissues with the clostridial bacilli (*C welchii* etc) cause extremely serious spreading infections which are cellulitic in character This group of microorganisms (Gram-positive anaerobes) normally inhabits the intestinal tract more commonly

animals than of man Wounds incurred here animal excreta is present (farm and eld) are presumptively contaminated These ganisms characteristically produce exotox s and their infections not only spread rap- ly but produce rapid gangrene of the host s ssue as well as gas. Such infections, there re are called generically *gas gangrene* he treatment of soft tissue wounds which e suspected of having gas gangrene infec ons is emergent Prophylactically one may elp to avoid such infections by the careful leansing and debridement of soft tissue ounds and the avoidance of devitalized tis ie In the definitive therapy the wound must e removed devitalized tissue must be cut way with sharp instruments, and the wound ust be left open to the air Fascial spaces ust be opened and muscles must be ex osed. The administration of specific gas angrene antitoxin (passive immunity) is nperative Systemic biotherapy is important ot so much because it is effective against the lostridia per se, but essentially because it revents the symbiosis of the clostridia with econdary contaminants (staphylococci and treptococci) The use in such wounds of aterials which produce nascent oxygen, uch as activated zinc peroxide, are no onger commonly used because the wound s now opened widely to the air while the djacent tissues are protected by systemic iotherapy

The disease of *tetanus* (lockjaw) is an nfection caused by a specific Gram positive naerobic spore bearing bacillus The organ- sm is a saprophyte and its normal habitus s in animal feces therefore it is found on he soil, particularly of farms. The portal of entry is any wound or break in the skin Tetanus infection would undoubtedly occur nore commonly if it were not for the fact hat the organism is a fastidious grower re- quiring anaerobic conditions in order to lourish. Its exotoxin is neurotropic in na- ture The incubation period is from 1 to 14 days however some cases of latent tetanus are known where the incubation period may be much longer Symptoms produced by this neurotropic exotoxin are essentially those of muscle spasm there is a fairly characteristic sequence of involvement of muscles the progression being from the mus- cles of the face and neck to those of the larynx and later other muscles are involved in a cephalad to caudad direction Spasm of the muscles of the face and neck gives rise to the characteristic expression the *risus sardonicus* of a patient with this disease The muscle spasms may be severe and painful and are terrifying to the patient, who is conscious throughout and endures this great mental agony Treatment of tetanus must al- ways be primarily prophylactic. It will be avoided by two main considerations first, the correct treatment of wounds (outlined in Chapter 3) second the immunization of the public at large with tetanus toxoid (active immunity) In addition following the in fliction of a wound a tetanus toxoid booster shot must be administered to patients who have been immunized and a prophylactic dose of antitoxin given to those who have not been The marked reduction in incidence of tetanus in military wounds incurred dur- ing World War II compared with those of World War I was due largely to routine active immunization of troops with tetanus toxoid Active treatment involves primarily the use of huge doses of tetanus antitoxin (passive immunity) Since the antitoxin is produced in equine serum caution must be exerted lest severe anaphylactic reactions oc- cur In addition, the wound responsible for the introduction of the spore-bearer into the host must be reopened and re-treated ac- cording to the proper principles of surgical therapy outlined in Chapter 3 Lastly bio- therapy must be employed in order to avoid the ravages of superimposed secondary in fection with common contaminants Mor- phine must not be used for the pain of tetanus, lest respiratory depression be in- duced External stimuli such as noise, light, and motion must be avoided since they tend to precipitate painful spasms of the involved

Specific Pathogens and the Diseases They Produce

TABLE 1

	Microorganism	Tissues involved and disease entity	Antibiotics and other useful agents	Remarks
Gram-positive cocci	Hemolytic streptococci	Respiratory tract (pneumonitis) Soft tissues (cellulitis) Joint spaces (suppurative arthritis)	Sulfadiazine Streptomycin Broad-spectrum preparations	
	Nonhemolytic streptococci	Respiratory tract Gastrointestinal tract in symbiosis with *E coli*	Sulfonamides and streptomycin	
	S aureus	Skin and bone	Penicillin systemically Bacitracin, topically Broad-spectrum preparations Albamycin, erythromycin	
	S albus	As above, mild Stitch abscess		
	Pneumococcus	Respiratory tract	Penicillin Broad-spectrum preparations Type-specific sera	
Gram positive coccus	Gonococcus	Urogenital, acute	Penicillin	
Gram-positive bacilli	*Mycobacterium tuberculosis*	Pulmonary Joints and bone Urologic Meninges	Streptomycin Isoniazid PAS	
	Corynebacterium diphtheriae and diphtheroids	Upper respiratory		
	Bacillus subtilis (spore-bearer)	Cutaneous wounds, burns Skin grafts		
Spirochetes	*Treponema pallidum*	Syphilis perivascular	Penicillin Heavy metals	
	Other spirilla	Mouth and gastrointestinal tract Vincent's angina Ludwig's angina	Penicillin Broad-spectrum preparations	

TABLE 1 (*continued*)

	Microorganism	Tissues involved and disease entity	Antibiotics and other useful agents	Remarks
am-positive bacilli	*E coli*	Intestinal tract, normal Peritonitis Ascending urologic Septicemia	Soluble sulfonamides Streptomycin for symbiotics Neomycin	All produce penicillinase and are therefore resistant to penicillin
	Salmonella typhosa and *S paratyphosa*	Intestinal tract	Chloramphenicol Neomycin	
	Pseudomonas procyanea	Cutaneous wounds burns skin grafts necrotic material	Bacitracin and polymyxin B topically Acid environment	
	Proteus vulgaris	Intestinal tract, burns Genitourinary tract Skin grafts, gangrenous tissue	Bacitracin and polymyxin B topically Acid environment	
	Nonhemolytic streptococci	Mouth and gastro-intestinal tract	Sulfonamide and streptomycin	
naerobic Gram-positive cocci	*Cl. welchii*	Devitalized tissue especially muscle, war and farm wounds fractures	Specific antitoxin versus specific exotoxin Open to air Activated ZnO, Penicillin	
	Tetanus spore bearer	Gastrointestinal tract of animals more than of man Wounds Selective affinity for nerves (neurotropic)	Specific antitoxin Irradiation Toxoid immunization	
Microaerophilic	Hemolytic and nonhemolytic streptococci	Gastrointestinal tract In symbiosis with staphylococci chronic (Meleney) Synergistic progressive gangrene	Penicillin and streptomycin Bacitracin and polymyxin B topically	

TABLE 1 (*continued*)

	Microorganism	Tissues involved and disease entity	Antibiotics and other useful agents	Remarks
Fungi	*Actinomyces* (sulfur ray)	Actinomycosis, lumpy jaw of animals Pulmonary bone Pulmonary tissues and bone, cutaneous sinuses and fistulae	Iodides CuSO Irradiation Surgical excision Broad-spectrum preparations for assorted infection	Actinomycosis is a fungus disease of *farm animals and* man due to the ray fungus *A bovis* It is characterized clinically by an acute and chronic infection resulting in formation of granulomas, sinuses, and fistulae. *Th human* portals of entry are a break in the skin ingestion into the alimentary tract, or inhalation into the respiratory tract. Although any tissue *may be the site of* a granuloma, the most commonly involved are face and neck lungs and thoracic cage, and the ileocecal area. Diagnosis is assured by microscopic identification Although iodides, sulfonamides, and irradiation have been helpful in therapy surgical excision is superior if feasible.
	Blastomyces	Blastomycosis (skin anus, pulmonary)	Do	*Blastomycosis is a* chronic cutaneous infection occurring usually near the anus. The granuloma resembles cutaneous tuberculosis and *advances* with a raised border Diagnosis is by microscopic identification Iodides are helpful in therapy

TABLE 1 (*continued*)

	Microorganism	Tissues involved and disease entity	Antibiotics and other useful agents	Remarks
Fungi	*Coccidioides*	Coccidiomycosis (pulmonary osseous cutaneous)		Coccidiomycosis is an acute and chronic granulomatous disease endemic to the San Joaquin Valley It may be cutaneous, respiratory osseous or generalized. The fungus is inhaled by man. Human-to-human and animal-to-animal infection is not known to occur Lesions resemble those of tuberculosis. Diagnosis is by microscopic demonstration of the endosporulating organism Complement-fixation serum studies are helpful.
	Sporotrichum	Sporotrichosis (animal excreta) Venous thrombosis and perivenous abscesses, cutaneous	Iodides	
Virus diseases	Surgical virology has not yet attained even the age of infancy The recognition, definition and culture of viruses are currently being pursued in many laboratories throughout the world. Surely the medical world has become more aware of human disease produced by viruses since the common use of biotherapeutic and chemotherapeutic agents has attenuated and changed bacterial diseases so greatly What role surgical therapy will play in virus-produced diseases, such as infections of the liver and of nervous tissue, remains to be determined. One virus disease which comes to the attention of the surgeon is rabies. Rabies is caused by a filterable virus which is neurotropic in character Negri inclusion bodies can be demonstrated in the brain of the infected host. The habitus of this filterable virus is in lower animals. Untreated, the disease is uniformly fatal The incubation period is from 21 days to as long as 6 months. The disease is usually transmitted from lower animals to the human being by means of a bite wound. The outstanding symptoms are nervous excitation and muscular spasm. Even minor muscular effort results in an aggravation of the spasms of the muscles, particularly of the larynx. Because of this, patients fear even the swallowing of water The name *hydrophobia* therefore became attached to this disease Prophylactically the disease may be avoided by accurate and rapid debridement of bite wounds. Whereas fuming nitric acid has been placed in such wounds in the past, it is now doubtful whether it has any advantages over rapid and thorough cleansing with soap and water The definitive therapy of the disease involves the use of antirabies vaccine—the Pasteur treatment.			

muscles Sedatives are most useful particularly antispasmotics and muscle relaxants, such as those of the curare group and succinylcholine

Infections of Bone

The pathogenesis of acute osteomyelitis is either (1) exogenous, caused by traumatic introduction of infectious material, e g a compound fracture or (2) endogenous, i e, acute hematogenous pyogenic osteomyelitis. In the latter instance one invokes the principle of focus of infection (Billings) In that the infection of the bone marrow cavity is *usually metastatic from another area*, near or remote The organism involved in acute hematogenous pyogenic osteomyelitis is almost always *S aureus* The pathogenesis involves the following sequence introduction into the medullary cavity of sepsis via mycotic embolus (hematogenous spread of bacteria) the extension of the infection from the medulla through cancellous bone the elevation of the periosteum by pus, and the extension of the infection into adjacent and overlying soft tissue The accumulation of necrotic material as a result of the osteomyelitis is called a *sequestrum* in that it has *become sequestrated or separated from normal tissue* The envelopment of the sequestrum by new bone laid down by neoperiosteal activity is referred to as the *involucrum*

The diagnosis is made on the history of the characteristic symptoms and signs *wherein the patient (usually a child) experiences* a tender and painful area along the metaphysis of a long bone not uncommonly following trauma in the vicinity Local and systemic signs of infection are to be found Within 2 weeks after the process has begun there will have been sufficient destruction of bone to produce a relatively radiolucent area (*sequestrum*) on the roentgenogram and thus reveal the diagnosis Differential diagnosis must include *acute suppurative arthritis*, tuberculosis of the joint, acute rheumatic fever and diffuse cellulitis.

Treatment of acute hematogenous pyogenic osteomyelitis in the prebiotherapeutic period was almost exclusively operative by early excision The sequestrum of the bone *was uncovered and removed the wound was* allowed to granulate in from below Acute osteomyelitis is far less common today when systemic biotherapy is so commonly employed for peripheral infections of both minor and major nature It can almost be said that the occurrence of acute osteo*myelitis today is consequent upon the neg*lected treatment of some form of peripheral infection

The term *chronic osteomyelitis* refers to the smoldering character of some infections of bone which do not become violent enough to elevate the periosteum and point toward the skin, but rather remain within the confines of the bone without ever being com*pletely* overcome by the defensive mechanisms of the body Such a diagnosis is occasionally made on a routine roentgenographic finding of a radiolucency the differential diagnosis of this disease in its chronic state (*Brodie s abscess*) *includes all* bone tumors and infections It is only fair to say that chronic osteomyelitis is the result of undiscovered or inadequately treated acute osteomyelitis The treatment today is surgical evacuation of the abscess cavity with adjunctive use of biotherapy

SUMMARY OF PRINCIPLES

1 The discipline of microbiology is basic to an *understanding of surgical pathology* and necessary in effective surgical therapeutics

2 The human host may be *contaminated by microorganisms* introduced through the skin or through other natural portals of entry

3 *Every episode of contamination* does not result in an infection of the host.

4 The transition from contamination to infection depends upon the element of time the severity and duration of the period of exposure and the relationship between virulence of the microorganism and resistance of the host

5 The rituals of the operating room are designed to avoid provision of new portals of entry for pathogens into the patient s body

6 Postoperative infections are most commonly of the operative wound of the respiratory tract and of the urogenital tract Reduction in incidence of postoperative respiratory and urologic infections can be accomplished by preservation of normal function of these systems.

7 Infections of the serous cavities which are closed spaces with broad absorptive surfaces, are governed by similar principles of therapy In addition the therapy of each is governed by factors peculiar to its anatomy and physiology

8 There are several types of soft tissue infections depending upon the nature of the invading microorganism and the response of the host s tissues to the invader Cellulitis is a diffuse spreading infection in which operative therapy is contraindicated and in which systemic biotherapy plays the major role There are several types of abscesses, each with its characteristic findings which determine the therapy in each case

9 Successful management of infections of the complicated structures of fingers and hands, with preservation of function depends on meticulous and detailed adherence to principles

10 Tendon sheath and fascial space infections must be detected and drained early at preferred sites of incision

11 Infections with clostridial organisms produce the most severe cellulitis with the production of exotoxins Prophylaxis of these infections is important, and active therapy is emergent.

12 Acute and chronic osteomyelitis are infections of bone, amenable to combined operative and systemic therapy

13 Reference is made to the table on specific pathogens and the diseases which they produce (Table 1)

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3

The Management of Wounds

I Definition and types
II Significance
III Management
A First Aid (1) hemorrhage (2) shock (3) contamination
B Definitive (1) the skin (2) muscles
IV Complications infection and associated injuries
V Special tissue injuries
A Peripheral nerves (1) etiology (2) pathology (3) symptoms and signs (4) management
B Tendons
C Blood vessels

DEFINITION AND TYPES

In the broadest sense a wound may be defined as any injury or damage to tissue with or without obvious solution of continuity A bruise a contusion and a subcutaneous hematoma are wounds in a very real sense of the word since damaged tissues must now be restored to normal yet there is no obvious solution of continuity There is actual destruction of cells within the thickness of the skin and in various layers underneath however which will evoke the inflammatory reaction and ultimately be healed with restoration to or toward normal

Open wounds have been the concern and for millennia the principal responsibility of the surgeon. These wounds include an infinite variety of types and a wide range of severity and importance from trivial cuts or scratches which never come to the attention of a physician to fatal injuries, with death resulting from hemorrhage shock destruction of vital organs or overwhelming infection. Although many of the specific types of wounds and their management and complications are discussed elsewhere in detail they are mentioned here for the sake of completeness The important common denominator in all open wounds, whether produced by violence or by a purposeful incision is the breach in the overlying integument whether it be skin or mucous surface The interruption in continuity of the protective barrier exposes the deeper tissues to contamination by pathogenic microorganisms, and thus to infection

Several types of wounds are described the nature of which determines their significance and management Among these are the *Incised wound* which is made by a sharp instrument such as the surgeon s scalpel or sometimes by a blow against a hard surface, such as the hard plate of the skull. The edges in an incised wound are sharp and clean, and there is a minimum of tissue destruction *Lacerated or torn wounds* such as those produced by jagged glass or metal or from crushing injuries, usually result in considerable tissue destruction. *Contused wounds* are

characterized by the crushing and devitalization of the tissues about the wound *Penetrating and perforating wounds* are those made usually by a narrow or pointed weapon or *by gunshot* Their significance lies in the fact that they produce a communication between the deeper tissues and the outside world, permitting the introduction of anaerobic organisms into the depths of the wound, and in the disparity between the visible wound and the possible extensive injury to deeper structures

The principles for successful management are common to all types of wounds in addition, special forms of wounds and injuries of certain structures require specific measures These include thermal injuries and wounds of bone (fractures) tendons nerves and larger blood vessels This also applies to wounds of the body cavities. These special considerations will be taken up separately for each of these injuries those of thermal injuries and of bone and blood vessels in separate chapters, and those of nerves and tendons in this chapter

SIGNIFICANCE

The extent and location of wounds cover an enormous range which affects the treatment and outcome The management of wounds of the integument is particularly important because the skin is the body s first line of defense against invasion by microorganisms with which one comes into daily contact throughout life Solution of continuity of the skin may also have an undesirable esthetic effect, which can be minimized with proper wound management This applies particularly to wounds of the hands fingers, and face which may have undesirable esthetic results and may also impair the ability of the patient to earn a livelihood or to follow a profession These undesirable results must be held to the irreducible minimum Wounds of special tissues which maintain normal body structure (bone) and of nerves and muscles which lead to the performance of specific movements are of special significance Compound fractur sent bones injured in open wound tration into or perforation of th cavities, chest, abdomen and skull tendant visceral injuries and expo infection all require special consic In the management of such lesion concerned not only with the princ therapy which apply to all wounds with those which will permit the rec tion of these special tissues so that th mal functions may again be carried as great a degree as possible

For optimal results of wound n ment it is imperative that the initia ment by the first doctor to see the be consistent with all well-established ples. Emergency rooms must be ade equipped and staffed by competent nel deviations from and indifference cepted principles at this time may c mise the ultimate recovery from wo all types Meticulous emergency roor apy will result in the saving of many and limbs and in the preservation of c function

MANAGEMENT

First Aid

Wounds vary as has been said o enormous range in extent, severity damage to important structures. Their agement, obviously is determined by factors. The first-aid phase of wound ment aims to prevent death from h rhage asphyxia, or shock to protec exposed surfaces from bacterial conta tion and to avoid further injury durin transport of the patient. Hemorrhage is ally amenable to control by compressio bandaging applied directly to the w Only occasionally when there is sever terial bleeding from the depths of a w which cannot be stemmed by compres is the tourniquet desirable Treatmer shock should be initiated as early as pos by the infusion of blood, plasma, or pl

expanders. Gentle handling the avoidance of all unnecessary movements, and the administration of narcotics contribute toward the prevention of shock The treatment of these life threatening complications must be continued throughout the course of the patient s management.

Definitive

The over all aims of wound therapy are the restitution to or toward pre-existing normal, of anatomic and functional integrity To accomplish this major aim one must (1) prevent soiling of the wound (implantation of bacteria) the multiplication of bacteria in the wound and the spread of infection to other areas (2) aid and abet the natural processes of wound healing (3) provide for systemic factors conducive to wound healing. Prevention of soiling of the wound depends upon adequate cleansing of the injured tissue and of the skin around it. Appropriate antitoxins or toxoids must be administered where applicable The systemic use of biotherapeutic agents should be undertaken judiciously In general it may be stated that measures which prevent contamination and infection also aid and abet good wound healing The latter include the removal of devitalized crushed and necrotic tissue, as well as the removal of foreign bodies Hemostasis must be accurate and yet one must exert caution not to devascularize the area unnecessarily Wherever questionably viable tissue remains or where there is excessive dead space, there must be provision for drainage Wound healing will be further enhanced by the correction of the following systemic derangements hypoproteinemia, hypohemoglobinemia, hypogammaglobulinemia and low vitamin C level

Cleansing of the wound mechanically is the initial step in the operative management of an accidental wound The wound edges should be cleansed with a bland soap and water or saline solution deep tissues may be irrigated with the sterile salt solution Cutaneous antiseptics should not be poured into the wound. The process of wound cleansing includes the mechanical removal of dirt and detached tissues as well as foreign bodies Zealous scrubbing of open tissues is not advisable lest the trauma already inflicted be compounded

Debridement the cutting away of devitalized and potentially nonviable tissue including the injured cells along the margin of the lacerated wound is next in order The term *debridement* is synonymous with *excision of the wound* This procedure is accomplished by sharp dissection, care being taken to remove all tissue which is separated from its blood supply but equal care being exerted not to sacrifice normal tissues particularly nerves, blood vessels, and tendons which may usefully be reconstructed in the future Some type of anesthesia, often local is usually required if debridement is at all extensive

The early and vigorous treatment of shock takes precedence over local care of the wound The treatment of bleeding from a very large vessel is therefore the initial concern of the first doctor to see the patient. In the case of injury to major vessels such treatment must always include an attempt to save the vessel by reconstructive suture or immediate graft replacement.

If the wound was sustained under conditions that increase the likelihood of anaerobic infections (farm, street, known contamination with intestinal contents) and particularly if there is much contusion and devitalization of muscle, the nonviable tissue must be cut away and the overlying fascial sheaths allowed to remain open. Anaerobic infection must be guarded against, also in penetrating and perforating injuries, since conditions for anaerobic growth are best in the depths of the wound

Closure of the wound by sutures should be carried out if the wound is treated within a short period of time after its infliction, if cleansing and debridement appear successfully to have removed grossly contaminating material, if anaerobic contamination is

not seriously considered and if, by judgment on the basis of past experience one may expect healing by first intention Should these factors not obtain, particularly if anaerobic infection is suspected, *the wound must* be left open and secondary closure attempted at a later date Under such conditions Vaseline *gauze may be placed in* the wound or provision made for continuous sterile saline irrigations occasionally irrigation with Dakin s solution (sodium hypochlorite) may be useful to remove necrotic material which cannot safely be cut away mechanically Enzymatic debridement may be accom plished also by some of the newer fibrinoly sins When wounds are closed per primam every attempt should be made to approxi mate tissues to a condition as near normal as possible This will permit better wound healing and will yield a better cosmetic result *Approximating stitches in the skin* should be of as fine material as possible under no tension, so that strangulation of tissue is avoided and should be as accurately applied as possible Particular attention in these regards should be paid to wounds of the face and of the hands

While the operative part of the wound management is being carried out, one is mindful of the systemic facets of wound management. These include the administration of a tetanus toxoid booster where the patient has had active tetanus immunity previously conferred or the giving of a test dose and administration of tetanus antitoxin (passive immunity) where active immunity does not exist. Gas gangrene antitoxin can be given during this time according to the assessment of the nature of the wound and *the conditions under which it was inflicted.* Blood transfusions can be ordered and ad ministered during this time if blood loss has been severe Pain medication is ordered as necessary Antibiotics should be adminis tered systemically

Immobilization of the wound structure is an important phase of the operative therapy of wounds this permits better healing of injured tissues and insures greater freedom from pain The type of immobilization will, of course, vary with the area of the body involved.

COMPLICATIONS: INFECTION AND ASSOCIATED INJURIES

The complications of wounds include pyogenic and anaerobic infections. The latter are essentially tetanus and gas gangrene The management of such infections begins logically with their prophylaxis, which includes the measures alluded to in the paragraphs above relative to wound management, and the systemic use of antibiotics and specific antitoxins Other complications are injuries to the deeper visceral structures, such as nerves, tendons and blood vessels The man agement of each of these will be discussed in detail. A poor anatomic result in the re *construction of a wound is regrettable* a poor functional result is deplorable Less than optimal results from a functional and esthetic point of view may compromise the ability of the patient to earn a living or to pursue his profession.

SPECIAL TISSUE INJURIES

Peripheral Nerves

Etiology Peripheral nerves are commonly injured by accidental wounds through exter nal violence, inadvertently during operations, or by the application of an appliance such as a brace. Nerves may also be injured by the displaced fragments of broken bones. Nerves most frequently injured by casts and braces are the radial nerve at the midhu merus level and the common peroneal nerve at the level of the fibular head below the knee (Peripheral nerves may also be injured by abnormal structures and spontaneous disease Examples are (1) the pressure of a cervical rib upon a branch of the cervical plexus, (2) aneurysms, (3) callus forma tion and (4) malignant tumors)

Pathology A peripheral nerve carries the lower neurone of the two-neurone system.

When a lower neurone is severed the distal fragment undergoes Wallerian degeneration (atrophy of the axis cylinder) regeneration of the neurone takes place from the proximal fragment, with the fatty nerve sheath acting as a directing conduit A neuroma is formed at the end of a cut nerve In a partial injury, such as a crush, a neuroma in continuity is formed This structure consisting largely of fibrous tissue, helps to provide the matrix through which the new neurone will grow Occasionally the fibrous tissue of the neuroma may be so large that it may interfere mechanically with regeneration from the proximal end

Symptoms and Signs. When a peripheral nerve is severed there is specific loss of function of the muscles innervated and anesthesia of the skin areas supplied. The paralysis is flaccid in nature (The electric findings known as *the reactions of degeneration* are clinically measurable this refers to the loss of faradic excitability in association with a sluggish reaction and later loss of response to the galvanic current, particularly the cathode closing current.) Since peripheral nerves are mixed in character there is a sensory loss corresponding to the cutaneous distribution. Loss of secretory function and trophic changes in the skin also may be observed, since the mixed peripheral nerve contains both motor and sensory fibers and also motor autonomic nerve elements to cutaneous glandular structures and to blood vessels. Inability to perceive noxious sensory stimuli may result in ulceration of skin Partial severance of a peripheral nerve as in crush injuries, may eventuate in the incompletely understood clinical condition known as *causalgia*. This entity is characterized by a markedly increased sensitivity to peripheral stimuli even light touch produces excruciating pain. The pain is described as of burning character and is so severe that the extremity is tenderly protected by the patient. The skin of the involved area, usually the digits, is moist and livid The complexity of this condition is attested to by the fact that sympathetic denervation usually corrects the moistness of the skin but only partially relieves the burning pain

Of particular importance is the diagnosis of injuries of nerves to the fingers hand and forearm because of the artistic, esthetic and economic importance of these structures Severance of the radial nerve in the forearm produces a paralysis of the extensors of the wrist (wrist drop) and of the extensor function of the proximal phalanges of the fingers and the thumb Some extensor function of the distal phalanges of the fingers is preserved through action of the interossei musculature Injury of the radial nerve where it lies directly over the middle of the shaft of the humerus (Saturday night palsy) will produce a paralysis of the brachioradialis muscle as well If the radial nerve is injured at its origin in the brachial plexus, paralysis of the triceps muscles also occurs Division of the median nerve on the volar aspect of the wrist, where it lies directly dorsal to the tendon of the palmaris longus produces the characteristic paralysis of the opponens pollicis, of the flexor indicis and flexor digitorum, of the flexor pollicis longus, and of the abductor pollicis longus. This *motor loss* includes the inability of the patient to appose the thumb to the other fingers, particularly to the fifth finger There is sensory loss on the radial side of the palm and of the distal two phalanges on the dorsal surface (Fig 3) Injury to the ulnar nerve at the wrist is also common it results in the loss of most fine movements of the fingers. All the interossei and all lumbricals save two are innervated by the ulnar nerve The fingers are extended at the metacarpophalangeal joints because of unopposed extensor (*radial*) action this plus flexion at the interphalangeal joints makes for the *claw hand* Adduction of the thumb is also impaired (Fig 4)

Management The treatment of peripheral nerve injuries in incised wounds varies with the nature of the wound and of the associated injuries. It adheres to the principles of good

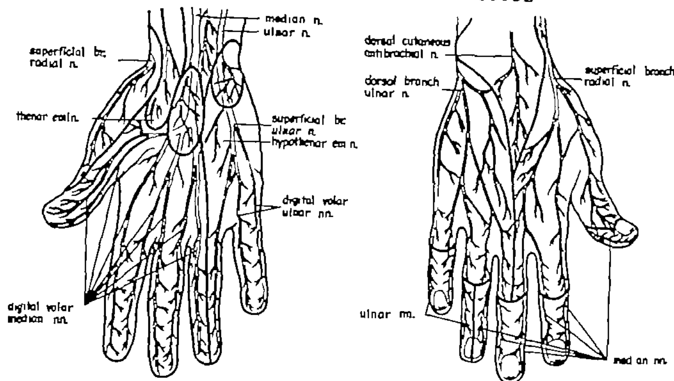


FIG 3 *A* Palmar *B* dorsal and digital cutaneous sensory nerve supply

wound treatment and requires, in addition the application of principles specific to nerve tissue Primary nerve suture, the repair of a nerve at the time of initial wound management, is permissible only when treatment is applied very early after the injury has been incurred when the wound is relatively clean, and when there is no gross comminution of tissues. When such conditions obtain, the ends of the severed nerve must be identified, sharply freshened, and then apposed with out placing sutures through the body of the nerve

If the nature of the wound is such as to preclude closure and healing by first intent, if several hours have elapsed since infliction of the wound and if there is much destruc tion of surrounding tissue then it is better to perform delayed suture of the peripheral nerve Under such conditions the ends of the nerve are identified by marking with nonabsorbable sutures for later use and the wound is treated according to the principles of good surgical practice previously outlined the nerve may then be sutured at a later date usually 6 to 8 weeks later at which time it may be accomplished under more optimal conditions At that time the edges will again be identified and their ends cut sharply and then apposed with nonabsorbable sutures.

It is possible, either at the time of initial wound therapy or at the time of delayed nerve suture, that loss of tissue will not permit the approximation of the two ends of the nerve Under such conditions a nerve graft may be placed and sutured between the widely separated ends for this purpose a cutaneous nerve of lesser importance may be used The graft serves only the purpose of a conduit, through which the growth of the new axone will be directed Postopera tive care is important and must include ade quate immobilization with minimal tension upon the suture line with the affected muscles splinted in position of function Since a period of time varying from 6 to 18 months may be required for the complete regenera tion of a peripheral nerve physiotherapeutic measures must be employed to maintain the vitality of the muscles, temporarily deprived of innervation. The skin served sensorily by the affected nerve must be protected from noxious stimuli which it cannot appreciate

Nerve injuries produced by disease may be reversible such as those produced by

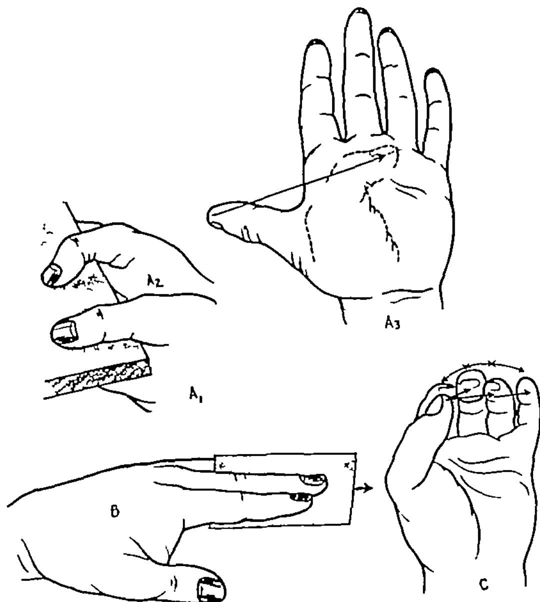


FIG. 4 Test for function of ulnar nerve. A_1 Normal hand grasps board, adductor pollicis holds thumb flat against board. A_2, Paralysis of adductor pollicis distal phalanx of thumb is flexed, and thumb cannot be flattened against board. A_3 Normal function of adductor pollicis. *B* Normally innervated interossei permit holding of a card tightly between fingers. If fingers are paralyzed, there is no resistance to pulling card away Additionally if the ulner nerve is paralyzed the distal and middle phalanges of the fingers cannot be extended. (Extension of the proximal phalanges is under control of the radial nerve.) *C* Test for median nerve function by combined action of abductor pollicis brevis and opponens, permitting combined opposition of thumb to fingers.

cervical rib and by vascular aneurysms, both of which lend themselves to surgical correction. When peripheral nerves are involved by malignant tumors, the latter are usually metastatic and very extensive multiple lesions are seen in the disease entity *neurofibromatosis* (von Recklinghausen s disease) In operations upon the parotid gland it may become necessary to sacrifice a portion of the facial nerve it is appropriate to replace the sacrificed portion with a nerve graft.

Tendons

Tendons are most frequently injured in the forearm wrist, hand and fingers where they perform the most artistic, esthetic, and economically useful functions. The flexor and extensor tendons of the wrist and hand

are commonly involved in lacerations on the volar and dorsal surfaces of the wrists, respectively These injuries are often improperly sutured under the poor conditions of many hospital emergency rooms It must be emphasized that tendon repairs are major procedures and should be done under the very best aseptic operating conditions in formal operating rooms. The timing of tendon repairs follows the same principles as those of nerve repairs, i e., primary tendon repair should be done only when the initial treatment can be carried out soon after infliction of the wound when the wound can be well cleansed, and when there is no great comminution of tissues and major associated injuries A primary tendon suture should never be performed in a wound which can not be adequately cleansed under such conditions it is much wiser to label the ends of the lacerated tendon and to affect a late repair, 6 to 8 weeks after the occurrence of the wound.

Repair of the deep flexors of the fingers is imperative the superficial flexors may be sacrificed and used as grafts to complete the repair of the deep tendons. Tendon repair must be anatomically precise and for this purpose it is recommended that an anatomy atlas be available in the operating room at the time of repair The tendons of the flexors of the fingers differ importantly from those of the extensors The former lie within firm fibrous sheaths for part of their course and have synovial investments to permit smooth gliding function. When divided, they retract widely being attached only at their insertions Furthermore, the distal fragment receives but meager blood supply from its bony attachment and from a tiny vessel in its mesentery (mesotendon) When injured, inflamed or sutured there is very great likelihood of crippling adherence of the tendon to the immobile and unyielding sheath. All these factors make for poor results unless the most meticulous methods and technics are employed The extensor tendons, by contrast, lie in loose areolar tissue (paratendon) which provides vascular supply prevents retraction, and interferes much less with subsequent mobility

In delayed tendon suture the cicatrix at the ends of the lacerated tendon must be sharply cut away before the ends are apposed by nonabsorbable sutures An attempt should be made to reconstruct the sliding mechanism so essential to smooth function of the tendon, particularly of the digital flexors. Loose areolar tissue of the forearm may be grafted and used for this purpose Tendon grafts may be used where there is loss of tissue in important tendons the graft may be taken from less important tendons, such as the palmaris longus in the forearm or the dorsiflexors of the fifth toe In the postoperative period immobilization must be adequate with minimal tension on the sutured site and the extremity in the position of function (Fig 5) To provide for maximal

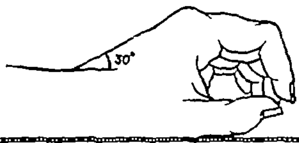


FIG 5 Position of function optimal position for ankylosis of wrist.

function and minimal synechias to adjacent tissues, active but not passive motion should be started as soon as the tendons are reasonably well healed usually about 3 weeks after suture Antibiotics should be used systemically particularly when primary suture is done this is important in tendon injuries, because if infection does take place the subsequent necrosis of tendon will lead to loss of tissue and the necessity for a graft at the time of secondary operation

Blood Vessels

Unlike nerve and tendon injuries, major arterial lacerations demand prompt and definitive management, because the life of

the limb depends on the early restoration of blood flow In terms of modern vascular surgery ligation of major vessels is not only unnecessary but intolerable After control of the proximal end of the vessel for purposes of hemostasis, the ends of a completely or partially divided arterial trunk must be identified properly prepared and sutured If there is loss of substance precluding the apposition of the severed ends of the artery a graft must be made as part of the initial therapy Suitable replacements include a portion of autologous vein of suitable size (with the direction of the vein reversed so that the valves will not impede the arterial flow) a homologous artery, or an inanim prosthesis

When an artery is lacerated by either complete transection or a partial section t artery distal to the injury undergoes mark spasm owing to an increased tonus of t sympathetic nerve supply to the arter vasculature Release of this sympathetic h pertonus is essential to the success of t arterial restitution This is accomplish both by the injection of papaverine into t adventitia of the vessel distal to its sutu and by surgical sympathectomy These mea ures are discussed in detail in Chapters and 11

SUMMARY OF PRINCIPLES

1 Wounds may be inflicted upon any tissues of the body including the integument and its derivatives muscles nerves tendons blood vessels and bone the viscera in the several body cavities may also be affected

2 Wounds are of many types including incised, lacerated contused, penetrating and perforating

3 There are basic principles for the successful management of open wounds of all types some of the special structures mentioned have special principles and technics which apply additionally to them.

4 Open wounds of the integument breach the body s first line of defense against infection to the integument itself and the deep tissues it covers

5 The over all aims of wound therapy are the restitution to normal of anatomic and functional integrity and prevention of infection following the initial contamination

6 Definitive management of an open wound includes cleansing debridement hemostasis precise reapposition of tissues and immobilization of the part.

7 Wounds which are potentially contaminated with anaerobes and in which there is much muscle comminution must not be closed per primam secondary closure at a later date is much safer

8 Attention must be directed also to the systemic aspects of wound management i e appropriate drugs to combat anaerobic infection, particularly tetanus and gas gangrene antibiotics and attention to protein and hemoglobin levels.

9 Particular attention must be paid to open wounds of those parts which perform esthetic and artistic functions particularly the hands and fingers

10 Peripheral nerves may be injured by external violence surgical appliances the healing of bone and malignant disease.

11 Peripheral nerve injuries are detected by the motor, trophic and sensory changes which are produced

12 The characteristic disability of injury to the major nerves of the hand must be known to all who deal with traumatic wounds

13 Primary nerve suture is performed only under the most advantageous conditions if they do not exist at the time of injury closure of the wound and secondary repair of the nerve at a later date is preferable.

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4

Shock

I Definition
II. Differentiation from simulated states: syncope and coma
III Theories of pathogenesis
  A Regional fluid loss and acute toxemia
  B Hepatorenal humoral
  C Infection
IV Reversibility versus irreversibility
V Pathology
VI Clinical etiology
  A Hemorrhage
  B Trauma
  C Burns
  D Infection
  E Pancreatitis
  F Other causes
VII Treatment
  A Prophylactic
  B Definitive

DEFINITION

Shock is a state of hypovolemia—decreased effective circulating volume It is characterized clinically by a lowered arterial blood pressure a rapid thready pulse a small pulse volume an ashen-cyanotic color a moist, cool, clammy skin thirst muscular weakness tachypnea a dulled sensorium and restlessness.

DIFFERENTIATION FROM SIMULATED STATES SYNCOPE AND COMA

The definition of shock is complicated by the fact that other pathologic states which only simulate it clinically but are not shock, are frequently included as types of shock. *Syncope* or *fainting* for instance, is frequently considered to be a neurogenic shock. It may be due to sudden emotional reflexes with or without a sudden fall in blood pressure It may be caused by vasomotor lability triggered by sensory stimuli or subjective thoughts hysteria, or sudden but temporary cerebral anemia. The cardinal manifestations of reduced blood pressure and tachycardia are either not present or of short duration owing to restorative cardiovascular nervous reflexes brought into play *Coma* or *neuroplegia* a state of unconsciousness at various levels, also only resembles shock but does not present its cardinal manifestations It may be due to cerebral vascular disease or injury or to an altered blood chemistry as in diabetes

THEORIES OF PATHOGENESIS

Regional Fluid Loss and Acute Toxemia

As defined above the essence of shock is a reduced effective circulating blood volume The most obvious explanation for the hypovolemia is actual blood loss to the out side Equally understandable is the loss of blood or plasma into traumatized tissues, organs, or body cavities or into areas in volved in and adjacent to burns and infarcted tissues Also reasonably understand able is the removal of large quantities of blood from the active effective circulation by profound vasodilatation such as occurs in anaphylaxis and with profound neurogenic disturbances which lead to preferential pooling of large portions of the circulating blood in one area as they are diverted from other areas (hematometakinesia) Marked reduc tion in cardiac output may also produce a reduced effective circulating volume causes include coronary artery heart disease cardiac trauma, and toxic myocarditis More difficult to understand is the increased capillary permeability with extravasation of fluids remote from the area of immediate trauma Moon at first ascribed this to the anoxic effects of the hypotension. Phemister believed chemical agents released by the traumatized tissues caused generalized vasodilatation and capillary hyperpermeability The toxic materials are proteins in various stages of breakdown. Although the theories of regional fluid loss and absorption of toxic products can account for many observed phenomena in shock, they fail to explain other observations

Hepatorenal Humoral

More recently a hepatorenal humoral theory has been elaborated (Shorr and Zweifach) which not only explains more of the observed phenomena in shock but also clarifies the pathogenesis and the intractability of shock produced by modalities other than direct blood or fluid loss These authors have demonstrated the presence in the blood stream of vasodepressor and vasoexcitor materials Vasodepressor material (VDM) is elaborated by the liver, it is a vasodilator and hypotensive Vasoexcitor material (VEM) is produced by the kidney it is a vasoconstrictor and antagonistic to VDM These materials apparently act upon pre capillary muscular sphincters, which are anatomically located between the metarterioles and the capillaries. VEM is believed to potentiate the action on precapillary sphincters of materials normally found in the circulating blood produced by the adrenal me dulla and at autonomic neuromuscular junc tions (sympathin E and sympathin I) VDM prevents the action of these latter materials upon the precapillary sphincters.

Infection

Recent studies (Fine et al.) have revealed the existence of a synergistic interaction of hypotension and sepsis. Hypotension impairs the native ability of the blood stream to clear itself of circulating pathogenic microorgan isms Conversely overwhelming infection induces hypotension The coexistence of the two lethal factors is important in the persist ence of shock despite adequate replacement therapy It is well known for example that in a period of profound hypotension, absorption of coliform bacilli through the in testinal mucosa into the blood stream takes place These organisms are a part of the normal flora of the intestinal tract and yet are not absorbed in health.

REVERSIBILITY VERSUS IRREVERSIBILITY

Irreversibility of the manifestations of shock after apparently adequate replacement therapy has been a puzzling and disconcert ing problem Were this understood therapy would be more logical and effective It has long been thought that shock becomes ir reversible when certain vital organs, such as kidney liver adrenals, and brain have been profoundly anoxic for a sufficiently long t me The VDM VEM humoral theory appears to explain, better than any other the

failure of some cases of hypovolemic shock to recover with apparently adequate blood volume restitution There is as yet no understanding of the factors evoking the continued elaboration and action of VDM by the anoxic liver Further projected work in this field gives promise of providing fuller insight which will lead to a more logical and specific therapy of irreversible hypovolemic shock.

PATHOLOGY

Measurable changes occur in the vascular system and in the chemistry of the circulating blood in shock The effective circulating blood volume is reduced. This is due to four main factors (1) loss of whole blood from the vascular tree (2) loss of plasma through capillary walls into the interstitial spaces (3) visceral engorgement with blood as a result of vasodilatation and reduced blood velocity in small blood vessels (4) sludging of red cells in capillaries venules, and arterioles. The hematocrit level does not necessarily reflect the state of the blood volume Immediately following hemorrhage the hematocrit is normal or only slightly diminished later after fluid shifts between the compartments have taken place the hematocrit will be markedly lowered. When the shock is due predominantly to loss of plasma from the vascular tree (burns) the hematocrit will be elevated early and will be reduced later by dilution as a result of intercompartmental fluid shifts Characteristic blood chemistry changes include (1) acidosis, due to excess accumulation of lactic, pyruvic and diacetic acids, which reduce the fixed base content of the blood, (2) elevation of urea, creatinine and nonprotein nitrogen The lowered blood pH is reflected by an increasingly acid urine, high in ammonia content.

Of prime and sometimes critical, importance is the diminished renal blood flow that occurs in shock. If the actual hypotensive state lasts sufficiently long oliguria or anuria may preclude recovery of the patient even though adequate restitution be made and blood hemogram and chemistries be corrected

CLINICAL ETIOLOGY

Hemorrhage

Hemorrhage the loss of blood from the vascular tree, is the most common cause of shock. Loss of circulating blood may be into tissue spaces body cavities, or the intestinal lumen or through the diseased or incised skin to the outside The pathogenetic sequence (as depicted in the unbalanced shock wheel) is as follows (1) diminished cardiac output (2) tissue anoxia (3) increased vessel permeability (4) further increased fluid loss into tissue spaces (5) diminished venous return

Trauma

Trauma has become an increasingly frequent cause of hypovolemic shock in this age of machines misunderstandings, and missiles Trauma includes the accidental crushing of tissues and the partially avoidable and partially unavoidable damage incurred during operative procedures. In addition to the relatively large amount of fluid loss into traumatized tissues, there is undoubtedly some absorption of partially broken-down protein material, which is toxic when absorbed into the venous tree.

Burns

In thermal burns, the ultimate in capillary permeability and loss of massive volumes of fluid into the tissue spaces occurs Geographically the areas of tissue that are inundated with plasma lost from the circulatory system are uniformly greater than would be expected from the observed surface area of the burn. For a detailed discussion of burns the reader is referred to Chapter 6

Infection

When extensive absorptive surfaces are exposed to infection the circulating blood

may be overwhelmed with bacterial toxins. These materials produce profound capillary permeability and fluid loss which leads to shock Examples of such massive infections are suppurative peritonitis and clostridial infections of large bodies of muscle

Pancreatitis

Acute necrotizing and hemorrhagic pancreatitis is characterized by shock out of all proportion to other clinical findings. This is attributable both to hemorrhage and to massive fluid loss following the unbridled action of the activated pancreatic enzymes within the peritoneal cavity

Other Causes

Profound shifts of fluid within the vascular tree may occur into a given area when the normal tonus of the blood vessels in that area is reflexively disturbed A frequent example of this is the profound vasodilatation of the splanchnic vasculature when the celiac ganglia are maximally stimulated, as by prolonged surgical traction at laparotomy or *after sustaining a blow to the intact abdomen* (solar plexus blow)

Anaphylaxis may be accompanied by profound and diffuse vasodilatation sufficient to produce a marked decrease in the effective circulating volume

TREATMENT

Prophylactic

The modern surgeon attempts to prevent the development of shock wherever possible in preference to correcting it after it has developed The following measures are of capital importance in the prophylaxis of shock (1) proper preoperative preparation of the patient for elective surgery such preoperative care includes the correction of (*a*) dehydration (*b*) hypoproteinemia, (*c*) depletion of glycogen stores and (*d*) hypohemoglobinemia (2) accurate hemostasis during operative intervention (3) gentle *handling of tissues* (4) *splinting* of extremities for transportation following accidental trauma (5) proper selection of anesthetic agents and (6) proper timing of the elective operation.

Definitive

The *restoration of effective circulating blood volume* as quickly as possible is of prime and critical importance This is best accomplished by the transfusion of fresh whole blood All other measures are secondary It is desirable not to wait until the hematocrit and hemoglobin concentrations are reduced by dilution The hematocrit, pulse and blood pressure levels may be criteria for restoration toward normal as therapy is being applied Certain blood substitutes may be utilized until compatible blood is available. The most useful of these is human plasma, which will help restore intravascular pressure relations but does not contribute to oxygen-carrying capacity Human serum albumin is also efficacious but does not contribute to oxygen-carrying capacity it is a very expensive product. Plasma volume expanders have become available in recent years The most commonly employed of these is a solution of 6 per cent dextran It has an average molecular weight similar to that of human serum albumin and is retained in the circulating fluid long enough to combat hypotension (about one half of it remains in the circulation at the end of 12 hr) Hemorrhagic tendencies may follow the use of massive quantities of this material Polyvinylpyrrolidone (PVP) is a synthetic material manufactured from acetylene and formaldehyde which is a good plasma volume expander About a third of the material is not excreted but remains trapped indefinitely in the liver presumably by macrophages Numerous gelatin products have been and are being further processed to make them useful in the treatment of shock It is important to point out that the plasma volume expanders (free of red blood cells) merely expand the circulating volume to correct one spoke of the unbalanced shock

wheel the diminished cardiac output and diminished venous return to the heart The control of hemorrhage and the replacement of red blood cells must follow their use as soon as possible Reduction of the duration of the hypotensive phase of hypovolemic shock to an irreducible minimum is of prime importance in the prevention of renal failure

Adjunctive measures to the definitive treatment of shock include the administration of morphine or its equivalent to promote physical rest and allay mental anxiety and the administration of oxygen by any suitable technic to increase the oxygen-carrying capacity of the remaining circulating blood It must be emphasized that the use of vasoconstrictor drugs is of only very temporary value and does not diminish the emergent necessity for restoration of effective circulating volume

SUMMARY OF PRINCIPLES

1 The cardinal and pivotal factor in hypovolemic shock is a diminished effective circulating volume.

2 The classic clinical picture is a patient with a lowered arterial blood pressure a fast thready pulse a small pulse volume an ashen-cyanotic color a moist cool clammy skin thirst muscular weakness a dulled sensorium and restlessness

3 Syncope or fainting is usually reflex in nature, it is of short duration, and the circulating volume is normal Although sometimes called *neurogenic shock* it is not true shock Similarly coma, or neuroplegia is not shock.

4 Suggested theories of pathogenesis of shock other than loss of blood to the outside, include (*a*) regional fluid loss, (*b*) absorption of toxic products (*c*) hepatorenal humoral factors (VDM VEM) (*d*) massive infection

5 Reversibility or irreversibility of the process of shock is probably the result of anoxia of several key tissues particularly the liver by which VDM is elaborated, the kidney from which VEM is produced and the adrenal cortex

6 The VDM VEM relationship probably acts by inhibition of and by sensitization of the metarteriole-capillary system respectively to substances elaborated by the adrenal medulla and the automatic nervous system

7 The blood in shock is acidotic owing to the reduction of fixed base by metabolic acids. This lowered blood pH is reflected in an acid urine with a high ammonia content.

8 The most frequent clinical causes of hypovolemic shock are (*a*) gross hemorrhage into tissue fluids, body cavities, or intestinal lumen, to the outside of the body or into infarcted tissues (*b*) trauma accidental or surgical (*c*) burns (*d*) massive infection (*e*) pancreatitis (*f*) anaphylaxis

9 The prophylactic therapy of shock, in so far as it is applicable is essentially the accurate preoperative preparation of patients for elective surgery This includes the correction of dehydration hypoproteinemia, hypohemoglobinemia, and depleted glycogen stores. During the operative procedure accurate hemostasis and gentle handling of tissues are desirable The

splinting of traumatized extremities for purposes of transportation is necessary Proper timing of an elective procedure and proper selection of anesthetic agents are helpful adjuncts

10 The definitive treatment of hypovolemic shock is the restoration of effective circulating volume preferably with whole blood

11 Plasma, plasma volume expanders and human serum albumin may be used as blood substitutes until compatible blood is available

12 Pressor substances are of temporary and limited value and are to be used only until definitive volume restitution can be made

13 The administration of oxygen, by suitable means is of aid in increasing the oxygen-carrying capacity of the diminished circulating volume

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5

Parenteral Alimentation; Fluid-Electrolyte and Acid-Base Balance, Protein Metabolism and Nutrition

I **Introduction**

II **Normal dynamics**

A Water balance

B Electrolyte balance

C Acid base balance (1) normal values (2) alkalosis respiratory and metabolic (3) acidosis respiratory and metabolic

III **Abnormal dynamics with normal kidneys**

A Dehydration with predominant water depletion

B Dehydration with predominant salt depletion

C Gastrointestinal losses severe combined depletions

IV **Abnormal dynamics with impaired kidneys**

A Anuria and oliguria

B The postoperative period the reaction of stress

C Crush syndrome

V **Nutrition**

A Protein dynamics (1) normal (2) in debility and repair (3) causes of hypoproteinemia (4) significance of hypoproteinemia

B Caloric requirements

C Supplementary factors

INTRODUCTION

The past quarter century has seen rapid advances *in technical* surgery which have extended the range and advantages of surgical intervention to patients with diseases heretofore considered beyond treatment. So great has been the progress that no organ or system is beyond the reach of the surgeon s beneficent attack. It must not be presumed *that these advances* are due to greater technical skill of the surgeon of today as compared with such titans of the past as Kocher Dupuytren, Mott, Billroth and Halsted, for such is not the case It is abundantly evident that these technical advances are consequent *upon a fuller* appreciation of basic physiologic principles and their practical applications in the care of patients. The *physiology* of the dynamics of circulation and *respiration*, of normal constituents of blood, *of* blood *substitutes, of normal* and abnormal renal function, and of nutrition in all its phases—all these are the foundation of modern surgery Knowledge of all these factors is essential to the trained surgeon for he must not only treat the *diseased* organ, but provide for the entire body economy For these *reasons the* surgeon s work begins much before the patient comes to the operat

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fluid) is detected clinically in the form of pulmonary edema and by venous distention By the time dehydration is recognized clinically by skin changes, dryness of the tongue and sunken eyeballs (*hypovolemia*) the patient has already lost 6 per cent of his body weight as water

Electrolyte Balance

The normal electrolyte pattern consists of a predominance of the sodium and the chloride ions. The chloride ion is ubiquitous, whereas the sodium ion is the predominant cation in extracellular fluid and the potassium ion in intracellular fluid. The pH of the blood in health varies only slightly from 7 45 It is determined mainly by (1) the ratio of the concentrations of the bicarbonate ion (HCO_3^-) and of carbonic acid (H_2CO_3) (2) the relative proportions of the biphosphate ion (HPO_4^{--}) and the acid phosphate ion ($H_2PO_4^-$) and (3) the relative availability of chloride (Cl^-) ions and sodium (Na^+) ions (fixed base) The normal blood and urine findings are as follows

	Serum	*Urine*
Sodium	315–340 mg per 100 ml or 136–145 mEq/L	Same as serum
Potassium	16–22 mg per 100 ml or 3.5–5 mEq/L	150 mg per 100 ml or 33 mEq/L
Chloride (as NaCl)	570–620 mg per 100 ml or 96–105 mEq/L	Same as serum
CO	27 mM/L or 61% combining power	

From the figures it is apparent that the blood serum and urine concentrations of sodium and chloride in health, are almost identical One might assume from this fact that any excess in intake of these substances under such conditions would be quantitatively excreted in the urine Such is found to be the case In depletion of either substance, the deficient ion would be retained and any excessive amount administered excreted if sufficient water were available Potassium is found in greater concentration in the urine than in the blood this indicates that the blood potassium level is relatively constant and that the urinary excretion parallels the intake This, it must be emphasized, is true only in the presence of normal renal function

The metabolism of potassium the chief intracellular cation, requires special comment. *Only a small fraction of the body* potassium is found in the extracellular fluid, 4 to 5 mEq per liter Potassium is important in nerve and muscle physiology it is necessary for the utilization of administered proteins and for the accompaniment of glucose into the cell Tissue breakdown of both proteins and glycogen releases potassium into the extracellular fluid even with these increments, the total amount of potassium in the circulating fluid is very small compared with total body potassium The concentration of potassium in the circulating blood is important, since relatively minor deviations from the normal range (hypokalemia and hyperkalemia) produce lassitude anorexia, and changes in muscular irritability The most important muscle so affected is the myocardium abnormal serum potassium levels can produce changes in heart action which are detectable both clinically and electrocardiographically Severe hyperkalemia may result in cardiac standstill Inadequate potassium intake for more than a day or two will result in a lowered serum potassium level since excretion of potassium continues at virtually its normal rate this differs from the regulation of sodium where reduced sodium intake is compensated for by renal conservation of this cation With severe tissue breakdown, such as occurs in trauma or severe debility the circulating potassium concentration may be sharply *elevated by potassium* released from destroyed cells

Potassium depletion occurs under conditions other than diminished intake When there is a major loss of chloride there is an associated loss of potassium as well Other situations are starvation the postoperative

ing room and does not cease until long afterward Among the most basic and fundamental considerations in the care of patients is the knowledge of the normal maintenance requirements of the body of those substances which are important in the homeostasis of the internal environment and of their deviations from normal in disease

NORMAL DYNAMICS

Water Balance

The body is composed of 65 to 70 per cent water by weight The water is distributed into three compartments (1) intracellular (2) interstitial, and (3) circulating fluid, the latter two compartments constitute the extracellular fluid. In an average individual of 70 kg (154 lb) the relative distribution of water would be as in Figure 6

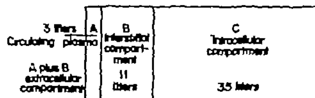


FIG 6. Normal distribution of water in a 70-kg individual.

Exogenous intake of fluid and the liquid content of solid food constitute the source of supply of 90 per cent of body water The remaining 10 per cent is accounted for by the endogenous water of oxidation The shift of water from chamber to chamber may rapidly alter the distribution of the total fluid content.

Loss of water from the body is by various routes and serves different functions

1 Loss from the skin (perspiration) serves the function of dissipation of heat
   a Visible changes readily with availability of supply and with external environment
   b Invisible constant within narrow limits and affected very little by exogenous supply varies from 1 000 to 1,500 ml daily
2 Urine provides a diluent for the excretable products of metabolism which average about 35 Gm daily For this purpose a urinary volume varying between 1 000 ml (at a specific gravity of about 1 020) to 1 500 ml (at a specific gravity of about 1 010) is necessary
3 Lungs vaporize 200 to 500 ml daily
4 Stool carries out 100 to 500 ml daily
5 An additional amount must be allowed for water loss on the following basis
   a Body temperature elevation, which increases visible perspiration.
   b Direct measurable fluid loss by vomiting, diarrhea fistulas, and gastrointestinal drainage

It will be noticed from Figure 6 that the intracellular compartment is by far the largest, constituting about 50 per cent of body weight the extracellular compartment, including both the interstitial fluid and the circulating plasma, comprises about 20 per cent of body weight. The boundary between the circulating plasma and the interstitial compartment is the capillary wall the boundary between the interstitial compartment and the intracellular compartment is the cell membrane The shift of fluid from one compartment to another proceeds at varying rates Fluid injected into the extracellular compartment may reach equilibrium with the intracellular compartment in a few hours equilibration between the circulating plasma and the interstitial compartment of course proceeds rapidly through the capillary wall

The ebb and flow of water between the circulating volume and the interstitial compartment is rapid and continuous. Fluctuations in the total amounts of extracellular fluid must be relatively wide before becoming clinically overt. Only after the changes have affected the intracellular compartment are the manifestations of edema or tissue dehydration clinically obvious *Hydremia* (excessive amount of circulating

fluid) is detected clinically in the form of pulmonary edema and by venous distention By the time dehydration is recognized clinically by skin changes, dryness of the tongue and sunken eyeballs (*hypovolemia*) the patient has already lost 6 per cent of his body weight as water

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Potassium depletion occurs under conditions other than diminished intake When there is a major loss of chloride there is an associated loss of potassium as well Other situations are starvation, the postoperative

period (partially due to the alarm reaction, since the excretion of potassium is to some degree controlled by the adrenal cortex) and, most strikingly bowel obstruction and/or ileus of long standing The clinical improvement in such patients following the administration of 40 mEq of potassium chloride (3 Gm KCl) is remarkable It may be given by the gastrointestinal route or parenterally

Acid Base Balance

Normal Values. There are a number of buffer systems operative in the body to maintain a relatively constant slight alkalinity of body fluids. At pH 7 the number of acid hydrogen ions is exactly balanced by the number of alkaline hydroxide ions Extracellular fluid is maintained between pH 7.35 and 7 45 The various buffer systems include the monosodium-disodium phosphate monopotassium-dipotassium phosphate oxyhemoglobin–reduced hemoglobin, plasma protein changes, and carbonic acid–sodium bicarbonate systems. The latter is the most important and can be used to illustrate the maintenance of acid-base homeostasis. Normally *there are 20 bicarbonate particles to* each carbonic acid particle At this ratio the extracellular fluid pH is 7.35 to 7 45 When

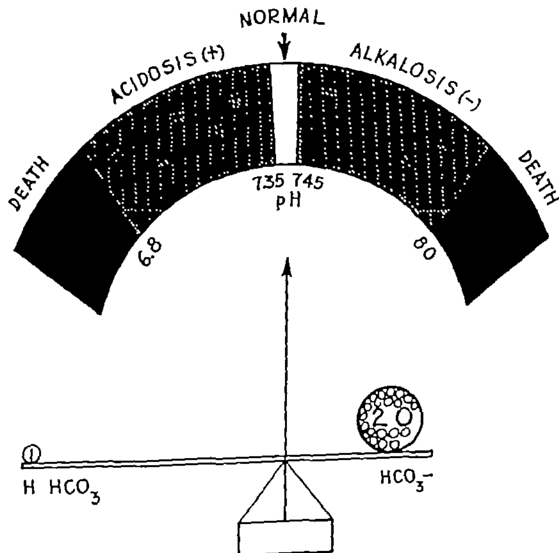
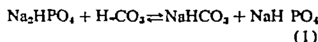


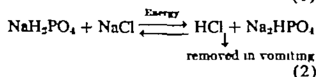
FIG 7 A diagrammatic representation of the Henderson Hasselbalch equation for maintenance of a normal blood pH by means of the buffer systems. A ratio of HCO_3^- H_2CO_3 of 20 1 maintains a normal pH of 7 35 to 7 45 an increased proportion (more than 20 1) of HCO_3^- (base) tends toward alkalosis a reduced proportion (less than 20 1) of HCO_3^- (base) tends toward acidosis

the HCO_3^- ratio is increased to 40 1 over the carbonic acid the pH approaches 7 8, dangerously alkaline Conversely when the HCO_3^- ratio is decreased to 8 1 or 10 1 over the carbonic acid moiety the extracellular fluid pH approaches 7 0 or even 6 8 dangerously acid. Figure 7 illustrates this phase of the Henderson Hasselbalch equation

Alkalosis, Respiratory and Metabolic. In *alkalosis* from any cause the pH of the blood, of course is elevated Deviations in this direction are brought about by two entirely different types of disturbance (1) *metabolic derangements* (the more frequent) and (2) *derangements of respiration* The most common cause of metabolic alkalosis is *vomiting* which results in a loss of chlorides from the stomach The formation of hydrochloric acid in the stomach and its loss through vomiting may be depicted as follows



$$Na_2HPO_4 + H_2CO_3 \rightleftharpoons NaHCO_3 + NaH_2PO_4 \quad (1)$$



$$NaH_2PO_4 + NaCl \underset{}{\overset{\text{Energy}}{\rightleftharpoons}} \underset{\substack{\downarrow \\ \text{removed in vomiting}}}{HCl} + Na_2HPO_4 \quad (2)$$

In order for the second reaction to proceed toward the right, energy is necessary since it results in the formation of a strong acid from its salt. This energy is provided by and is the secretory function of the acid producing cells of the gastric mucosa. When the HCl is removed by vomiting, there is an excess of fixed base (Na_2HPO_4 and the $NaHCO_3$) in the blood, constituting the *metabolic alkalosis* The correction of this condition is by replacement of chlorides in the form of sodium chloride and the kidneys can then excrete the excess sodium as $NaHCO_3$ It has only recently been appreciated that the replacement fluids should also contain some KCl, because with every chloride loss there is concomitant loss of intracellular potassium

Respiratory alkalosis is due to excessive elimination of carbon dioxide gas from the lungs This is seen clinically in *hyperventilation* which occurs (1) following head injury owing to stimulation of the medullary respiratory center (2) during improperly administered inhalation anesthesia or (3) in certain psychiatric disturbances in which hyperpnea occurs as an expression of the patient s anxiety The loss of carbon dioxide leaves an excess of bicarbonate ion, which combines not only with sodium but also with calcium The binding of the calcium ion lowers the circulating level of free ionic calcium and the clinical state of *tetany* results. This is manifested by increased neuromuscular irritability which may eventuate in convulsions

The correction of respiratory alkalosis is easily effected if its cause is recognized The measures used are (1) supplying CO_2 by inhalation or by simple rebreathing or (2) administration of calcium ion, preferably by the intravenous route

Acidosis, Respiratory and Metabolic Acidosis as the word implies is a disturbance in the opposite direction, in which the pH of the blood is depressed This too may be caused by either metabolic or respiratory derangements.

Metabolic acidosis takes two forms, *inorganic* and *organic* In inorganic acidosis there is an excess of serum chloride owing to loss of sodium The main causes are (1) diarrheas (2) intestinal fistulae, and (3) biliary fistulae The digestive fluids lost in these conditions are predominantly alkaline, and the depletion of the fixed base lowers the blood pH to the acidotic level Correction of this type of acidosis depends upon the replacement of fixed base with nonchloride solutions, such as sodium bicarbonate or sodium lactate These anions are metabolized or excreted, leaving the sodium to combine with the excess chloride thus raising the pH toward normal

Organic metabolic acidosis is a result of metabolic arrest, in which incomplete oxidation of carbohydrate occurs This is the clinical state of *ketosis* and is the metabolic disturbance of diabetes mellitus The lowering of the pH of the blood is a direct result

of the presence of acid ketone bodies—β hydroxybutyric acid, diacetic acid and acetone—which replace the HCO_3^- ion Proper treatment involves adequate replacement of base and glucose, with provision for more complete oxidation of the latter (in sulin) If, in addition to the organic acidosis (ketosis) there is water depletion inorganic alkalosis may be coexistent, owing to the inability of the kidneys to excrete sodium If so treatment requires all four elements water base chlorides, and glucose

Respiratory acidosis is due to an excessive amount of carbon dioxide in the blood This is primarily a ventilatory derangement in which, because of pulmonary pathology (emphysema) or hypoventilation (anesthesia) carbon dioxide cannot be eliminated from the alveoli of the lungs

ABNORMAL DYNAMICS WITH NORMAL KIDNEYS

The term *dehydration* has been loosely used in clinical parlance for the loss of body fluids. Such loss, however may consist of any one of the following (1) dehydration with predominant *water depletion* (2) dehydration with predominant *salt depletion* and (3) combined and complicated depletions such as occur with various gastrointestinal fluid losses

Dehydration with Predominant Water Depletion

This condition occurs particularly when water is purposely withheld or is unavailable or when a patient can ingest only an insufficient amount Under such conditions the extracellular compartment becomes temporarily hypertonic The body adjustments that take place to compensate for this altered state are the following (1) water flows from the intracellular compartment to the extracellular compartment (2) the urinary output is diminished This is represented diagrammatically in Figure 8 where it can be seen that the main contraction is of the intracellular compartment. The contraction of the circulating fluid volume results in the earliest finding of water depletion, a *low urine volume*

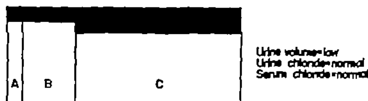


FIG 8 Water depletion.

Dehydration with Predominant Salt Depletion

When there is excessive chloride loss, as in profuse and protracted perspiration and in uncompensated loss of gastric fluids, the extracellular chamber becomes hypotonic. The body adjustment that takes place to compensate for this altered state consists essentially in excreting excess water without a corresponding amount of salt. This contraction of the extracellular compartment may become serious, because the reduction in circulating fluid volume (hypovolemia) is not compensated for by a shift of water from the intracellular compartment A sufficiently diminished circulating volume constitutes shock acute or chronic depending on the rapidity of the precipitating loss This is diagrammatically shown in Figure 9 under these conditions the kidney will "save" sodium and chloride, resulting in the earliest finding of predominant salt depletion, namely a *low urine sodium chloride content* The serum chloride will also be low when the salt depletion is great

Thus it is clear provided renal function is adequate that the urine will reflect these changes even before the circulating blood does In the case of water depletion there is a diminished urinary output and in the case

of salt depletion there is an abnormally low urine chloride concentration

A simple urine test suffices to measure the chloride concentration It is known as *the Fantus test* and is simple to perform It should be available at every hospital nursing division To gain the information desired one must know the urine volume which assesses the degree of hydration, and the concentration of chlorides in the voided specimen The normal urine chloride concentration is approximately 550 mg per 100 ml (5.5 Gm per liter) approximately the same as blood. With any finding of 3 Gm per liter (300 mg per 100 ml or 0 3 per cent) or more one may assume that there is no serious loss of chlorides. Under these circumstances one would start the correction of dehydration with glucose in water or with other solutions low in chloride content. If the urine chloride concentration has fallen below 3 Gm per liter (less than 0 3 per cent) there is definite indication for the early administration of saline solution.

The Fantus Test. The ingredients are (1) 20 per cent potassium chromate and (2) 2.9 per cent silver nitrate. One places in a test tube 10 drops of urine and 1 drop of the chromate which is an indicator only Then one places 1 drop at a time of silver nitrate in the tube, which is all the while being agitated, until there is a sudden color change from yellow to brick red. The number of drops of silver nitrate used to obtain this change is the number of grams of chloride per liter in the urine: thus 3 drops used to elicit the end point indicates a urine chloride concentration of 3 Gm per liter

If the urinary concentration is so low that it approaches zero one must then resort to serum chloride determinations, since such very low figures may be a result of renal disease or cardiac decompensation In patients with such complicating factors the use of clinical judgment is called upon (an appraisal of the hydration of the eyeballs, tongue skin etc) in the evaluation of the patient. It must be borne in mind that, by the time one recognizes well-established dehydration clinically the patient has already lost at least 6 per cent of his body weight. The urinary findings would have detected these deficiencies earlier

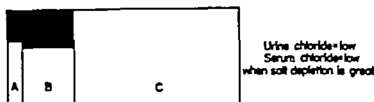


FIG. 9 Salt depletion.

Gastrointestinal Losses, Severe Combined Depletions

Body water is used by different organs and in different places in the body for different purposes. It carries some substances in solution and others in suspension and acts as a vehicle for the transport of many vital chemicals. Body water therefore is not the same thing at all places at all times it is one thing as a part of the blood, it is another thing as bile gastric juice pancreatic juice saliva, etc In its many forms and variations, the body water constitutes the *internal milieu* of the organism Losses of some body fluids and juices can therefore result in dehydration which is very complex. Correction of these complicated gastrointestinal fluid losses depends on an understanding of the basic physiology of the alimentary tract.

Parenteral alimentation is the modality of maintenance and therapy employed when nature's normal route the gastrointestinal tract, cannot be used for feeding as in the early postoperative period or in certain diseases of the gastrointestinal tract, with or without vomiting Knowledge of the normal physiology of the gastrointestinal tract in health and its pathologic physiology in disease is therefore inextricably bound to the principles of parenteral alimentation This is

readily understood when it is appreciated that the daily shifts of fluid from the circulating blood into the gastrointestinal lumen and back into the circulating blood is of the magnitude of two or more times the total circulating blood volume These large volumes of fluid carry water and enzymes into the alimentary tract for purposes of hydrolysis and digestion of foodstuffs and their assimilation back into the blood stream These materials are poured out mainly from the stomach duodenum and jejunum and are reabsorbed through the ileum and right colon. The nature of the various fluids depends on the order in which they serve in the intestinal tract.

TABLE 2

GASTROINTESTINAL FLUIDS

Fluid	Volume, ml	Reaction
Salivary glands	200– 300	Alkaline
Gastric juice	500– 1,500	Acid
Bile, from liver	500– 1,500	Alkaline
Pancreatic juice	500– 1 000	Alkaline
Succus entericus	1 000– 7 000	Alkaline
Total	2,700–11,300	

The normal shift of these fluids into the alimentary tract lumen and back into the circulating blood may be disturbed in disease *whether accompanied by* vomiting or not. Particularly in intestinal obstruction great losses may be sustained when these *fluids* are poured into the upper intestinal tract and cannot reach the lower intestinal tract to be resorbed. The fluid so accumulated *in the bowel lumen is as lost to the* body as if it had been spilled to its exterior These vital materials may also be lost *through purposeful gastric or intestinal suc*tion by fistulae accidental or purposeful (drainage of the common duct, ileostomy), *by vomiting or by diarrhea. Parenteral ther*apy must take into account not only the amount of fluid lost from these various *sources but their respective natures* as well.

Thus vomiting of essentially acid gastric contents will produce an alkalosis (elevated blood pH) owing to excessive loss of chloride ion *Replacement must be made with* isotonic or hypertonic chloride solutions. Fluids lost from the upper small intestine *include gastric juice bile* pancreatic juice, and succus entericus. All except the first of these are alkaline in character Replacement *of such losses is best accomplished* by a solution which is about 60 per cent sodium chloride and 40 per cent sodium lactate or bicarbonate the average daily requirement of 40 mEq of potassium ion must be included in replacement. Fluid and electrolyte loss in severe diarrheas is essentially of small intestinal fluids, high in fixed base. If not rapidly and adequately replaced, acidosis (reduced blood pH) will rapidly ensue Replacement requires fluids that readily provide fixed base, sodium bicarbonate or ⅙*M* sodium lactate In diabetic acidosis (ketosis, where the organic acids combine with fixed base) correction of the inorganic imbalance must be made with sodium bicarbonate or lactate while the metabolic error is appropriately simultaneously corrected.

ABNORMAL DYNAMICS WITH IMPAIRED KIDNEYS

Deficient renal function not only constitutes an additional hazard to the recovery of the surgical patient, but also greatly magnifies the difficulty of fluid and electrolyte control. Renal insufficiency may be on a *prerenal renal* or *postrenal* basis The coexistence of diseases such as glomerulonephritis or nephrosclerosis (renal basis) requires the cooperation of the medical colleague The presence of these diseases often precludes surgical intervention other than that which is emergent in character Reduced renal output on the basis of ureteral or prostatic obstruction (postrenal) demands their immediate correction by the appropriate urologic technic It is the prerenal causes of

excess of water is eliminated The salt lost in the urine during these 2 or 3 days must be replaced in part and by the oral route if possible

The Postoperative Period, the Reaction of Stress

As a part of the stress, or *alarm* reaction (Selye) there are certain characteristic changes in electrolyte control during the first 24 to 48 hr after an operation which have no relation to hypotension These include the inability of the kidney to excrete excess quantities of sodium or water and the simultaneous increased excretion of potassium This disturbance may be duplicated by the administration of adrenal steroids The surgeon should restrain the tendency to give too much fluid during this short period Also at this time, he should rely primarily on solutions of glucose and water with the addition of about 40 mEq per day of potassium If electrolytes are administered at this time it is preferred that they be given as half isotonic concentrations in 5 per cent glucose The early postoperative stress reaction is further characterized by a *negative nitrogen balance* which is due *to an increased excretion of* adrenal steroids. This process may be abetted by a *temporarily* increased excretion of L triiodothyronine a fraction of the thyroid hormone evidence is not conclusive

Crush Syndrome

A specific type of renal insufficiency is observed in extremely severe and widespread trauma which involves, particularly the skeletal muscles It is characterized by anuria or oliguria which are due to blocking of the renal tubules by masses of *myoglobin* pigment derived from the crushed muscle fibers Clinically this syndrome may be differentiated from the hypotensive lower nephron syndrome described above by the detection of myoglobin crystals in the urine by spectrophotometry It is also associated with marked hyperkalemia from the excess of potassium liberated from the destroyed muscle cells

NUTRITION

Protein Dynamics

Normal. Protein is the fundamental constituent of living tissue In the animal body it makes up the bulk of muscle and of visceral and epithelial structures, in the blood it constitutes many physiologically active factors The maintenance of normal protein metabolism for the exercise of vital functions is basic to good nutrition In surgical practice protein dynamics assume particular significance, because deviations from normal may preclude or delay recovery from otherwise remediable diseases. Patients who have withstood extensive surgical procedures but who still have serious deficiencies somewhere in the metabolic sequence of protein ingestion transportation, utilization, and storage may exhibit evidence of these deficiencies, such as anemia, cachexia, nutritional edema and indolent appearing wounds

In blood protein is found in plasma and in circulating cells the former essentially in *transit, the latter performing vital functions* Protein stores exist mainly in muscle *and in the liver*

The normal circulating plasma contains from 6 to 8 Gm of protein per 100 ml this represents a balance between supply storage and catabolism About two-thirds of this amount is albumin and one third globulin The albumin molecule is much the smaller of the two and is the important factor in maintenance of *oncotic* (*colloid osmotic*) *pressure* and the return of extracellular fluid to the capillary lumen Deficiency of protein therefore leading to edema, is due essentially to lack of the albumin fraction. The globulin fraction contains active and potentially active immunologic elements. Plasma contains other proteins, fibrinogen and prothrombin which are essential to the normal blood clotting mechanism they are precur

sors of fibrin Plasma from which they have been removed is serum

The main protein of the circulating cellular elements is the hemoglobin of the erythrocytes It is the essential element in oxygen transportation Some leukocytes plasma cells, and probably lymphocytes manufacture globulins which have immunologic properties.

In Debility and Repair During periods of increased catabolism circulating proteins are used to rebuild destroyed tissue, and they in turn are replaced by stored proteins When proteins in storage are depleted ingested protein will be utilized for reconstitution of body proteins, provided the patient has an adequate caloric intake If the caloric intake is not adequate ingested or parenterally administered amino acids will be used for conversion to energy If that situation exists, weight loss will be progressive In general, the state of the protein stores is reflected by the circulating protein concentration but this *is not an invariable relationship In certain* diseases such as multiple myeloma or sarcoidosis the fabrication of abnormal globulins in excessive quantities may produce plasma protein levels out of all proportion to the body stores.

Causes of Hypoproteinemia

1 Inability or failure to ingest
2 Impaired digestion and/or absorption
   a Chronic diarrheas (regional enteritis, malabsorption syndromes, such as sprue dysenteries, celiac disease)
   b Intestinal fistulae
   c Gastrointestinal siphonage
   d Intestinal obstruction (including ileus) *in which the intraluminal fluid* physiologically is lost to the body economy
3 *Inadequate synthesis* liver disease
4 Increased catabolism
   a Fever
   b Elevated BMR
   c Infections
   d Extensive malignant disease
5 *Increased protein loss*
   a Ascites
   b Nephrotic syndrome
   c Trauma, anesthesia, and anoxia
   d Hemorrhage
   e Burns

Significance of Hypoproteinemia. The range in total circulating protein concentration from normal levels to hypoproteinemic states, is narrow Relatively slight depres*sions may reflect significant disturbances.* The diminution by 1 Gm per 100 ml of plasma proteins means that 30 Gm of protein has disappeared from the circulation (The average adult has approximately 6 000 ml of blood, of which about 3 000 ml is plasma therefore 1 Gm per 100 ml diminution equals a total of 30 Gm of protein lost.) However the actual deficiency is much greater It has been shown (Pack and Rhoads) that before 1 Gm of protein disap*pears from the circulating fluid there has* been a depletion of 30 Gm of protein from the storage depots (liver and muscle) Therefore a drop of a single gram per 100 ml of circulating plasma proteins means that 30 × 30 or 900 Gm of *body* (storage) *proteins* have disappeared The weight loss as a result of these 2 lb of protein loss is aggravated by the energy utilized in the process of protein breakdown and an associated salt and water loss, all of which adds up to a loss in body weight of 6 to 10 lb This fact can almost invariably be confirmed by careful interrogation of the patient, unless the loss is *masked by concurrent water retention* (cardiac decompensation) or sodium retention (associated renal disease)

The most important protein in the body if such assessment can be made, is the special protein of the red blood cell, hemoglobin The hypoproteinemia of debility is almost always associated with a depletion of hemoglobin. When protein is administered in any such combined depletion the hemoglobin exercises priority its demands must

be met first before the level of circulating proteins begins to rise † It must be remembered that a normal total protein level does not exclude the possibility of hypoproteinemic edema, since elevation of the globulin may mask the albumin deficit This apparent contradiction is encountered in such situations as multiple myeloma, sarcoidosis, or certain forms of hepatic disease

Caloric Requirements

The need for an adequate caloric intake as an adjuvant to protein replenishment has been mentioned before It has been shown by detailed nitrogen metabolism studies that parenterally administered amino acids were utilized largely for energy requirements until sufficient calories were supplied to "spare" the proteins for their intended *anabolic* purpose Intravenous caloric input may be enhanced by the use of glucose invert sugar and alcohol and particularly by high caloric (9 Cal per Gm) fat emulsions Alcohol has the additional advantage of being an excellent sedative An effective blood level of 80 mg per 100 ml is reached by giving the first 200 ml of a 5 per cent or 7 5 per cent solution in the first 15 to 20 min (150 to 180 drops per minute) and then reducing the rate to 80 drops per minute

In estimating the caloric needs of the bedridden patient, the following factors must be taken into consideration The resting metabolic requirements of an adult average 25 Cal/kg/day For the usual activities incident to moving about in bed an additional 30 per cent of that figure is needed For each degree of fever another 8 per cent of the original figure must be added. Furthermore, if alcohol is used another 8 per cent must be added because of its stimulating effect on the metabolic rate Thus for a 70-kg patient, the caloric need is computed as follows

70 × 25 Cal =	1 750 Cal	(basal requirement)
30% of 1 750 Cal =	526 Cal	(bed activity)
16% of 1 750 Cal =	280 Cal	(2 deg of fever)
8% of 1 750 Cal =	140 Cal	(use of alcohol)
	2 695 Cal	total per day

While it may not always be important to prevent the patient from losing any weight, it must be pointed out that the approximation of his caloric needs is far from accomplished by the customary administration of some 2,000 ml of 5 per cent and 1 000 ml of 10 per cent glucose, which together supply only 800 Cal (200 Gm × 4 Cal) Even with the use of 10 per cent invert sugar (dextrose and levulose) and amino acids and alcohol, it is difficult to supply more than 1 500 Cal parenterally daily Recent trends toward the use of parenteral fat emulsions give significant promise Preparations now available supply 675 Cal per 500 ml of infusion With their aid the progression of acute hypoproteinemia to chronic protein deficiency with its resultant edema, impaired wound healing, lowered resistance to infection and further impairment of liver function will probably be prevented

Supplementary Factors

Since the potassium ion must accompany amino acids through the cell membrane if they are to be used as building blocks this cation must be administered together with parenterally administered protein Another useful adjunct to anabolism in the reconstitution of body protein stores is the use of testosterone and its related steroids These agents have been shown to promote healing

† The copper sulfate falling drop test may be used to assess approximately both values when a patient is seen for the first time, or it may be used to follow his progress from hour to hour or day to day provided that hydration is adequate. A serum specific gravity of 1 027 (or 1 056 for whole blood) roughly indicates a concentration of 7 Gm per 100 ml of protein and 15 Gm of hemoglobin. A deviation in the specific gravity of 0 0029 is equivalent to a change of 1 Gm per 100 ml in circulating plasma protein level

by enhancing protein synthesis their ability to do this is attested to by conversion of a negative to a positive nitrogen balance in states of excessive tissue breakdown

Finally attention must be given to the administration of vitamins especially the water-soluble B complex particularly necessary for hepatic and nervous system metabolism and vitamin C necessary for optimal wound healing The latter is not stored in the body and is heat-labile at febrile temperatures in vivo Vitamin K is added when specific need is present (liver disease and intestinal antisepsis.)

SUMMARY OF PRINCIPLES

1 Parenteral alimentation is the provision of nutrition to the patient by routes other than the alimentary tract usually intravenous. This includes (*a*) maintenance fluids electrolytes calories proteins consumed in daily catabolic processes and supplements such as vitamins and trace minerals and (*b*) replacement of all abnormal losses of body fluids electrolytes and proteins. Relatively rapid and reliable laboratory tests are available for the determinations of all these important components of body fluids

2 A knowledge of fluid-electrolyte and acid-base balance is essential to the effective use of parenteral alimentation.

3 Water and electrolytes are distributed into three different body compartments predominant water depletion and predominant salt depletion are differentiated, since they produce different shifts in the various components

4 Inorganic acidosis, the lowering of blood pH is a result of excessive loss of fixed base It is treated with alkaline fluids Inorganic alkalosis, an elevated blood pH is the result of excessive loss of chloride ion and an excess of fixed base. It is treated by replacement with chloride ion

5 Homeostatic maintenance of a relatively constant blood pH of about 7 45 is mediated through the bicarbonate and biphosphate buffer systems

6 Respiratory alkalosis is due to excessive blowing off of CO_2 from the lungs, as in hyperventilation, its correction involves the reversal of the cause of hyperventilation and the auxiliary use of intravenous calcium ion to prevent the tetany of hypocalcemia, since calcium ion is bound in alkalotic blood. Respiratory acidosis is due to excessive retention of CO_2 by the lungs and is corrected by artificial hyperventilation.

7 Potassium depletion occurs when oral intake is impossible the kidney does not save potassium as it does sodium under similar conditions

8 It is essential that the status of cardiac and of renal function be assessed when hydration and alimentation is effected by the parenteral route

9 The technic of parenteral alimentation is complicated in the presence of renal insufficiency Of particular interest to the surgeon are those periods of renal insufficiency known as *anuria* and *oliguria* (particularly of the prerenal variety due to hypovolemic shock) and the early postoperative period, in which there is a special form of oliguria related to the stress, or alarm reaction The crush syndrome is a specific type of renal insufficiency and is due to blockage of renal tubules by muscle pigment, myoglobin which is detected photometrically

10 During periods of anuria or oliguria no attempt may be made to force the kidneys to higher levels of excretion The patient must be kept "on the dry side"

11 In the early postoperative period associated with the stress reaction there is abnormal retention of sodium and water and excretion of potassium Sodium-containing fluids should not be administered at this time

12 In the presence of cardiac decompensation the body requirements of fluid and electrolytes may be altered These changes must be considered in planning the parenteral program

13 Protein must be included in parenteral therapy when it is needed to replace tissue proteins lost by basal catabolic requirements and in order to avoid a negative nitrogen balance Necessary to the effective use of administered proteins by the parenteral route are sufficient provision for caloric intake (lest amino acids be used for energy requirements) and potassium ion which must accompany amino acids into the cell

14 Hypohemoglobinemia is the most important type of hypoproteinemia

15 Total circulating serum protein level is a reflection of supply usage and storage of proteins Hypoproteinemia is accompanied by tissue edema, on an osmotic basis Laboratory values of total serum protein concentration are not significant without fractionation into the albumin and globulin components The level of total serum albumin concentration is the key to hypoproteinemic edema In certain forms of hepatic disease multiple myeloma and sarcoidosis there may be a hypoalbuminemia and hyperglobulinemia.

16 The causes of hypoproteinemia are inability to ingest proteins, impaired digestion and/or absorption, inadequate synthesis (liver disease) increased catabolism and increased protein loss

17 Modern surgery is based on physiologic principles. Intelligent use of parenteral alimentation in the surgical patient demands the understanding and application of the basic concepts of fluid-electrolyte and acid base balance nutrition in all its phases including protein carbohydrate and fat metabolism renal and cardiovascular respiratory physiology

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6

Thermal, Chemical, Electric, and Actinic (Radiation) Burns and Injuries

I Burns
- *A* Nature and complexity of the problems involved
- *B* Assessment of extent of wound
- *C* Pathologic physiology of burns (1) the local wound (2) systemic derangements (*a*) shock (*b*) infection (*c*) toxemia (*d*) anemia
- *D* Treatment of the local wound (1) initial (2) late
- *E* *Therapy of systemic derangements (1) treatment of shock* (2) treatment of infection (3) treatment of toxemia and anemia

II Cold injuries
- *A* Pathologic physiology (1) actual freezing (2) without freezing
- *B* Treatment

III. Chemical Injuries
- *A* Agents
- *B* *Pathology*
- *C* Treatment

IV Electric Injuries
- *A* Burn (1) character of local lesion (2) pathology and late complications
- *B* Shock (electrocution)

V Actinic (radiation) injuries
- *A* *Character of lesions*
- *B* Pathology
- *C* Complications
- *D* Treatment

BURNS

Nature and Complexity of the Problems Involved

The management of extensive burns is a twofold problem It includes the management of an open wound of a special type and the prevention and correction of severe physiologic derangements of basic systems The management of extensive burn injuries demands the precise application of knowledge in the following areas (1) the inflammatory process and factors involved in wound healing, (2) the prevention of contamination and the treatment of infection (3) shock (4) recognition and correction of fluid and electrolyte imbalance, (5) parenteral alimentation and nutrition. The consideration of all the factors involved in an extensive burn may tax the knowledge wisdom and patience of

the most mature surgeon and the best nursing personnel as well as fitting hospital facilities As in many other complex injuries and illnesses one learns that in the management of extensive burns one treats not just an injury but the entire patient These efforts are most often attended by success when there is adherence to fundamental surgical knowledge and principles and meticulous attention to details

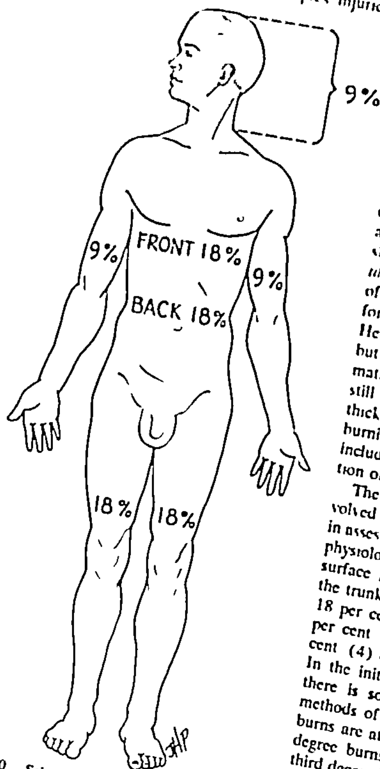


FIG 10 Scheme for estimation of body surface area involved in burns (the rule of multiples of 9) head and neck 9 per cent each upper extremity 9 per cent each lower extremity 18 per cent (9 per cent front, 9 per cent back) trunk 36 per cent (18 per cent front 18 per cent back)

Assessment of Extent of Wound

Burn wounds are classified according to depth or extent of body surface area involved A first degree burn is one in which only the superficial layers of the epidermis are involved an *erythema* is produced in the skin without evidence of vesiculation *Vesiculation* or blister formation is characteristic of second degree burns the burn fluid is formed between the dermis and epidermis Here there is destruction of the epidermis but healing may still occur without scar formation because the stratum germinativum is still intact In third degree burns the entire thickness of the skin is destroyed and the burning of tissue may extend to any depth, including charring of an entire limb or portion of the body

The extent of the body surface area involved by burn is an important consideration in assessing the degree of systemic pathologic physiology (Fig 10) In general the body surface is divided roughly as follows (1) the trunk 36 per cent (18 per cent ventral 18 per cent dorsal) (2) head and neck, 9 per cent (3) each upper extremity 9 per cent (4) each lower extremity 18 per cent In the initial assessment of a burn patient there is some overlap between these two methods of estimation, in that third degree burns are at least twice as severe as second degree burns thus, the derangements in a third degree burn of 20 per cent of the body surface are as severe as in a second degree burn of 40 per cent of the body surface

Pathologic Physiology of Burns

The Local Wound The disturbance of the normal body economy is initiated at the time and site of the burn and proceeds rapidly in the very early postinjury period The

cellular damage incurred by the burning agent is always more extensive than it at first appears to be There follows an increased blood supply to the area of the burn mediated through vasodilatation Profound increase in capillary permeability ensues and edema fluid pours into the tissues adjacent to the burn area and onto the burned surface *Edema fluid is essentially blood* without the cellular elements i e plasma.

Systemic Derangements. The results of the pathologic physiology of the wound are rapid, profound, and generalized The extensive outpouring of plasma into and around the burned tissues results in a diminished circulatory volume since the fluid that is extruded is essentially plasma a hemoconcentration rapidly follows The hypovolemia quickly results in a diminished cardiac output despite a compensatory tachycardia this rapidly eventuates in generalized tissue hypoxia, which is followed by metabolic acidosis and electrolyte imbalance One recognizes in this pathologic spiral the cardinal features of clinical *shock* which constitutes the first major problem with which the surgeon must deal in the therapy of burns

Any burn to the extent of second degree or worse is a wide open wound. This open wound allows the first line of the body's defense against infection to be breached Contamination by common pathogens is inevitable and is the rule plasma poured onto such wounds and the burn fluid in the vesicles are an ideal culture medium for these microorganisms Local and systemic *infection* then is the second major problem with which the surgeon deals in the therapy of burns.

Within a few days after occurrence of a severe burn *toxemia* is common This systemic debility is probably due to the absorption of intermediate products of tissue breakdown at the burn surface as well as of the bacterial exotoxins and endotoxins It is characterized by marked weakness, feeling of ill-being and prostration

A cytotoxic *anemia* occurs within several days to a week after extensive burn injuries (earlier in children and infants) This is presumably due to the above named toxins acting upon the erythrocytes which are hemolyzed

Treatment of the Local Wound

Therapy of the local wound will be considered first in order to preserve the line of reasoning in the development of the pathologic physiology of burn injuries However it must be pointed out that therapy of the local wound does not take precedence over the treatment of shock, which takes priority over all else The treatment of the local wound is begun concomitantly with the therapy of shock, or after shock has been successfully combatted

Initial. Burn surfaces are frequently contaminated by extraneous material such as clothing street dirt, oily substances, and obviously dead skin Although it is probably true that the more such dirty foreign, and dead tissue is removed the better will the remaining body defenses be able to cope with the problems of contamination and infection judgment must be exercised regarding how much time is spent accomplishing this end Too enthusiastic attempts at debridement may actually contribute to shock Time so spent must always be governed by the improvement in the state of shock which is brought about by simultaneous correction of the hypovolemia

In general necrotic tissue and grossly contaminated skin is cut away by sharp dissection Some surgeons prefer to open the vesicles and remove the overlying epidermis others prefer to leave unruptured vesicles intact. All surfaces should be cleansed very gently with bland soap and water (or saline) avoiding rough handling of these delicate injured tissues Cleansing and debridement may aggravate the patient's pain therefore sedation limited anesthesia and gentleness in carrying out these procedures are indicated

There are two principal methods of deal

ing with the burned surfaces The prevailing method is to attempt to convert the open wound into a closed one by immediate covering of the surfaces Recently the open treatment of burns has been revived and is finding considerable acceptance When covering is used the greatest variations in different hospital practices are in the selection of the initial layer There is general agreement that one layer of petrolatum gauze is probably as good as any other The dressing applied over the petrolatum gauze is a heavy fluffy one surrounded by a semielastic bandage for compression The purpose of the compression is to minimize the seepage of plasma from the wound and by immobilization through its splinting action to facilitate healing Such a dressing is particularly proper for the extremities occasionally for the trunk but hardly at all for the head and the genitalia

The painting or spraying with various dyes such as tannic acid, gentian violet or triple dyes, is seldom practiced at this time Particularly tannic acid is not used, since it is widely absorbed from the extensive surface and has when used in the past resulted in liver damage The other dyes are also not used because of their tendency to precipitate proteins and to cause the loculation of infectious material underneath the eschars which they form Similarly the use of picric acid is frowned upon because of the large number of persons who are sensitive to this substance The use of biotherapeutic and chemotherapeutic ointments on extensive burn surfaces, although used frequently in the past, is now discouraged this is particularly true of the sulfonamides, since they are rapidly absorbed and not infrequently lead to renal complications.

Those surgeons who use no dressing but rather leave the wound open, occasionally spray the wound with liquid paraffin, which has analgesic as well as covering properties they claim for it the same advantages formerly attributed to the use of the triple dyes, i e the formation of an eschar which allegedly prevents further seepage of fluid onto and from the wound However there is some evidence that the formation of eschars on burn surfaces does more harm than good in that it allows infection to become pocketed and retained rather than to drain freely onto the bandages

In summary with regard to the treatment of the local wound it may be said that gentle cleansing and sharp debridement are the first concern The use of dyes is more frowned upon than encouraged at the present time If a dressing is used the compression type should be used on the extremities Antibiotics must be administered systemically gas gangrene and tetanus antitoxin or toxoid are administered as for any extensive wound The area of the burn wound must be immobilized as much as possible to facilitate healing Ordinarily the first dressing is not removed for inspection for 7 to 10 days, unless complications are suspected as evidenced by continued high fever tachycardia or severe pain When the initial dressing is removed the areas of first and second degree burn are usually healed The deeper lesions require further treatment, with the objective of preparing them for definitive healing as early as possible

Late Subsequent management has for its purposes (1) the elimination of any surface infection (2) the completion of debridement (3) skin grafting to areas of third degree burn and (4) the prevention of contractures Whereas skin burned to a depth of second degree will heal without appreciable scarring, full thickness burns will be replaced by heavy and unsightly scars if the process of granulation is allowed to continue to its natural end To forestall such an undesirable result all areas burned to a depth of third degree are replaced by skin graft as soon as local and systemic factors are favorable There is no specific time in days for this surgical procedure but in general it may be done (1) when the infection of the wound appears to be bacteriologically controlled [late infections of wounds are caused by in addition to the usual pathogens which are ubiquitous, *Pseudomonas aeruginosa* (*pyocyaneus*), the

green-forming organism] and (2) when the systemic pathologic physiology has been sufficiently corrected to ensure good wound healing

In recent years there has been a tendency to replace areas burned to a depth of third degree with skin grafts earlier in the post injury period. Early resurfacing of extensively burned areas may be a trying and pressing problem particularly in children The sooner extensive third degree burns are closed with suitable coverings the sooner will *the loss of fluids proteins, and electrolytes* be diminished, the quicker will there be surcease from pain and the earlier will infection be brought under control and, with it the toxemia of bacterial endotoxins diminished If the area of third degree burn appears relatively clean after initial debridement (when the patient is first seen) skin graft may be done even at that time The skin grafts most commonly employed are autologous in nature and split thickness in character In extensive burns particularly in children there is often insufficient uninjured skin adequately to cover the denuded area Recently as a temporizing procedure *homologous* grafts have been found to be lifesaving It must be emphasized that, with *rare exceptions, such grafts cannot* be expected to survive permanently They may be completely and permanently successful only in identical twins and in the presence of agammaglobulinemia Cadaver skin is a convenient source for such grafts and the operation of skin banks (similar to blood and bone banks) for the taking and use of post mortem homografts appears to be a promising endeavor both for civilian use and for possible mass casualty considerations in the future

Therapy of Systemic Derangements

Treatment of Shock. The first threat to the patient's life—shock—must be combatted vigorously and promptly In the early hours and days of a burn a plan of management calculated on usual requirements must be followed later it must give way in many respects to individualization based on clinical judgment. The period of the danger of death from shock is from the time of burn to the end of the third day Even if the shock should be overcome the danger of resultant anuria is still a possibility if the initial period of hypotension was too protracted. As in every case of shock the cardinal principle of treatment is the restoration of the normal circulating volume In shock due to burns this is initially achieved by giving plasma *since this is the material which is lost* If plasma is not available in sufficient quantities plasma volume expanders such as dextran may be used as part of the requirement

A convenient and satisfactory plan for the early replacement of the deficient circulating volume is that of Evans. This calls for (1) about 2,000 ml of fluid per day to provide for the maintenance needs of renal excretion and insensible water loss from the skin and (2) 150 ml (about 1 ml of fluid per pound of body weight) per 1 per cent of body surface burned, for the first 24 hr The 150 ml is to be divided as follows one half (75 ml) plasma and one-half (75 ml) of an electrolyte solution similar to the composition of blood. Such an electrolyte solution may be one containing 0 6 per cent NaCl 0.3 per cent $NaHCO_3$ and 0 2 per cent KCl Of the combined total of plasma and electrolyte solution to be administered for the first 24 hr half of such volume is to be administered during the first 8 hr the remaining half is to be divided into one half for each of the next two 8 hr periods

Example for a 50 per cent burn in a 150-lb adult $50 \times 150 = 7{,}500$ ml this plus 2 000 ml of maintenance requirements = 9,500 ml for *the first 24 hr Of this total one-half* or 4 800 ml is to be administered during the first 8 hr period, 2 400 ml during the second 8-hr period, and 2,400 ml during the third 8-hr period.

The efficacy of this initial plan must be *followed by (1) the urinary output, as de*termined hourly by indwelling catheter (2) electrolyte and nitrogen balance studies and

(3) hematocrit and total circulating protein levels

If a normal urinary output of 30 to 50 ml per hour is maintained the plan of treatment will be judged to be adequate with regard to volume this will be confirmed by (1) the maintenance of a normal blood pressure and (2) the return of an elevated hematocrit reading toward normal If the hourly urinary output falls below 30 ml per hour either one of two things may be true (1) the amount of fluid administered is insufficient under which conditions increasing the amount of intravenous fluids will result in an increased urinary output (2) renal insufficiency exists and any increased amount of fluid administered will not increase the urinary output Thus a water load trial will serve to make the differential diagnosis between (1) and (2) above If the water load does not result in an increased urinary output no attempt must be made to increase further the amount of intravenously administered fluids since pulmonary edema may result To aid in avoiding this catastrophe auscultation of the lungs is performed at frequent intervals. After the initial few hours of this or any other plan of intravascular replacement of fluids the electrolyte pattern is followed carefully by blood studies. When the normal circulating volume has been restored whether during the first 24-hr period or at any time during the first 3 days diuresis will occur and must be carefully watched for

Careful laboratory determinations of all intake and output in the burned patient provide the best guide for the quantitation of electrolytes administered Accurate laboratory analysis of exact sodium potassium, and nitrogen intake and output will avoid wide deviations from normal particularly if alimentation is by the parenteral route When the burned patient can take food by mouth, the problems of fluid electrolyte and nitrogen metabolism are largely alleviated

There is no substitute for the laboratory determinations of total circulating proteins (and especially of the albumin fraction) and of the hematocrit and red blood cell count in determining the ability of the patient to rebuild destroyed tissue

Treatment of Infection There is hardly a surgical problem in which more extensive use of the principles and practice of surgical microbiology is made than in extensive burns The open burned wound bathed in the body s best nutritive material (plasma) is an ideal culture medium for the usual pathogens the staphylococci the streptococci and the colon paracolon group These agents are most successfully combatted by the use of biotherapeutic agents systemically administered penicillin is most effective against the staphylococci and soluble sulfonamide and streptomycin against the streptococci colon paracolon bacillus infection responds best in most cases to chloramphenicol Cultures should be taken in all cases and when the bacteria are identified sensitivity tests to the various drugs should be determined Death from infection usually occurs between the third and seventh days following the burn The most troublesome secondary contaminant to open wounds such as burns is the *Ps aeruginosa (pyocyaneus)* organism it is usually effectively controlled by the use of polymyxin B locally weak acetic acid mixtures are also effective in retarding such growth Urinary tract infections are common particularly with the colon paracolon group usually in patients in whom repeated urinary bladder catheterizations are necessary or in whom a retention catheter must be used Overgrowth of intestinal pathogens [particularly with *Staphylococcus pyogenes* and with *Ps aeruginosa (pyocyaneus)*] is not uncommon with extensive oral administration of broad-spectrum antibiotics Stool cultures must be made and therapy administered accordingly Intercurrent infections such as pneumonitis, are common

Treatment of Toxemia and Anemia Effective therapy of the toxemia and anemia which may result in death of the patient

from the seventh day on begins prophylactically with adequate therapy of the initial period of shock, the avoidance and control of the ensuing infection and the early recognition and correction of hypoproteinemia The toxemia probably depends largely on the products of tissue breakdown and on the absorption of the exotoxins and endotoxins of bacteria. Anemia due to red blood cell hemolysis becomes manifest in the adult from the fourth to the seventh day it occurs much earlier in the infant and in the young patient it is not illogical to use blood during the replacement therapy on the first day in children High-caloric and high-protein diets enable the body better to combat the toxemia, probably through improved hepatic function The sooner positive nitrogen balance is restored the sooner will toxemia be overcome Anabolic agents such as the androgens aid in saving nitrogen lost in tissue breakdown Successful closure of the local wounds with appropriate coverings limits the general malnutrition and debility During this time blood transfusions are of greatest value

COLD INJURIES

The parts of the body most commonly involved in thermal injuries by cold are those parts most exposed to the external environment the hands and fingers the feet and toes the nose ears and cheeks. These small structures all have a large surface area compared to the total volume

Pathologic Physiology

Actual Freezing There are two main types of thermal injury by cold and although the pathologic physiology in general is the same there are minor variations *Actual freezing* of the parts (frostbite) produces tissue damage very similar to that of burns. The intracellular water is converted into ice crystals and cellular death results Intense vasoconstriction of the adjacent arterioles results in the initial blanching of the part. In this stage there is anesthesia. Thawing is accompanied by a reactive hyperemia and exudation through the thermal-damaged capillaries With these changes appears severe pain of a deep burning character Vesiculation of the overlying integument supervenes and later separation of the necrotic portions occurs Infection or gangrene, of a dry or wet type may lead to loss of digits or limbs Healing is slow because of a tendency toward sustained vasoconstriction plus actual impairment of blood flow as a result of thromboses Prolonged hyperesthesia and sensitiveness, particularly to thermal stimuli is a distressing sequel which resembles causalgia

Without Freezing The second type of injury is referred to as *immersion foot* (or *immersion hand*) and results from long exposure to lowered temperatures particularly with high humidity and without actual freezing. The pathologic physiology may be traced as the symptoms progress At first there is vasoconstriction of the small parts with concomitant pallor then there is a reactive vasodilatation as evidenced by hyperemia and redness tissue edema due to increased capillary permeability follows the pressure of this excess fluid (the inflammatory exudate) is great enough to obstruct small blood vessels which then become thrombosed causing further tissue hypoxia and damage The thromboses are due to the deposition of fibrin and the sludging of cells in small vessels Secondary infection may supervene and further complicate the picture The end result may be a causalgialike picture with trophic changes of the finger tips or toes, including lividity marked hyperesthesia increased perspiration and burning pain

Treatment

The prophylaxis against thermal injuries by cold involves proper protection particularly of the fingers and toes by warm and especially dry clothing Gloves, shoes and boots impervious to moisture will protect against immersion hand or foot, which may

otherwise occur at temperatures above freezing

In the direct therapy of cold injuries it is best to avoid the use of rapid thawing by the application of heat and to avoid the opposite extreme of using cold as well The damage to tissues will be held to an irreducible minimum if the frozen part is allowed to thaw at room temperature The use of anticoagulants is logical and useful to avoid intravascular thrombosis as a result of fibrin deposition and sludging of cells Reflex vasodilatation of the part by the application of heat to other parts of the body is beneficial The logic of this procedure is similar to that of avoiding direct heat to a limb which has been made ischemic on the basis of arterial occlusion reflex vasodilatation is permissible but direct heat to the area would be damaging by increasing its metabolic demands for oxygen which cannot be met In a similar manner reflex vasodilatation by systemically administered drugs is also permissible Vasodilatation may also be accomplished by chemical sympathetic block or by peripheral nerve block In the extreme one may resort to surgical sympathectomy Adjunctive measures include the use of antibiotic ointments locally to help combat contamination and infection and the elevation of the part to aid venous and lymphatic drainage of the limb

CHEMICAL INJURIES

Contact of the skin or mucous membranes with corrosive chemicals constitutes a special type of burn injury The systemic pathologic physiology is identical with that of thermal injuries, except that it is rarely as extensive as is commonly seen in thermal burns Usually chemical injuries are much more sharply localized to small exposed areas of the skin Local pathology includes direct necrosis of cells including those of small blood vessels in the area Later the dead tissue is sequestrated from viable structures leaving an indolent ulcer Since ulcers are usually third degree in depth, repair is accomplished by the grafting of skin particularly if the area involved is large Therapy differs from that of thermal burns in that the initial treatment of the chemical burn varies according to the nature of the noxious agent

Agents

Acids and *alkalies* are the most common agents involved in chemical burns in civilian life Initial treatment involves the removal by dilution with running water weak opposites may be useful Corrosion of the pharynx esophagus and stomach are commonly due to the accidental or purposeful ingestion of strong acids and alkalies, particularly by children The therapy of esophageal strictures is discussed elsewhere

Pathology

Chemical burns with phenol (carbolic acid) are of a special type because this agent in dilute solution produces anesthesia in the skin and in strong concentration produces immediate coagulation Accordingly there is deep penetration of the noxious agent with ensuing thrombosis of small blood vessels Initial treatment of phenol burns is dilution with alcohol Phenol should not be used therapeutically on fingers toes or other thin parts because of its anesthetic properties Dry gangrene is the rule in strong phenol burns as a result of the almost immediate coagulation of tissue including blood vessels

Treatment

Most chemical burns occur as war injuries, occasionally as laboratory or home accidents Phosphorus burns are much less common in civilian life than they used to be because of precautionary measures used in industry The initial treatment of phosphorus burns involves the removal of particles under water in a dark room Dilution may be accomplished with alkalies A solution of 1 per cent copper sulfate may be applied to the skin with imbedded phosphorus particles the copper sulfate will react with the phos-

phorus to form soluble copper phosphate and insoluble cupric sulfide

Magnesium burns (the lesions caused by incendiary bombs) lead to very rapid ulceration of the skin. Initial debridement is essential in early therapy

The most common cutaneous vesicant is mustard which produces vesicles and ulceration Since this substance is oily it must be dabbed from the skin and not rubbed. Gasoline and other hydrocarbons are used to remove this oily substance from the skin they produce free chlorine gas Soap and water are second best in the initial treatment of this type of burn Lewisite is a gas the main ingredient of which is arsenic. Initial dilution must be made with hydrogen peroxide, alkalies soap and water and British anti-lewisite (BAL)

ELECTRIC INJURIES

Electric current passing through living tissues which offer resistance generates heat at the points of contact with the body and, additionally disturbs vital nervous centers on its way through the body

Burn

Character of Local Lesion The heat generated by the passage of such current through living tissue when properly controlled, may be used therapeutically as in medical or surgical diathermy Electric burns are produced at the points of entrance and exit when a current of sufficiently high voltage and low amperage passes through the body Alternating current more often causes serious burns than direct current The heat generated by a spark or gap of current, is very high and can produce very severe flash burns The local lesions may be round or oval a dirty gray or gray yellow they are usually sharply demarcated and do not produce vesicles there is little or no local pain Such burns are characteristically indolent i e they remain the same for long periods of time showing no tendency to heal then at a later date, when they might otherwise have been expected to be healed, they slough even further and more widely The final slough is always larger than the original burn

Pathology and Late Complications. The local pathology of the burn lesion is essentially a disintegration of the media of the arteries in the area this is in addition to the actual destruction of skin that takes place at the time of the burn. The disintegration of the media of the vessels accounts for the indolence and for the late complications infection and secondary hemorrhage The treatment of the local lesions in electric burns is identical to that of thermal burns, with a watchful eye for the late complications mentioned.

Shock (Electrocution)

The systemic pathology in electric shock or electrocution is either (1) the immediate production of ventricular fibrillation of the heart by direct action on its conduction system or (2) direct paralysis of the respiratory center

ACTINIC (RADIATION) INJURIES

Radiation injuries are a subject of increasing interest and concern to the peoples of the civilized (?) world, not alone because of the threat of mass casualties by atomic missiles but because of the widespread use of sources of radiation in industry and in medicine Occupational hazards to medical personnel and industrial technicians have increased multifold during the past decade

Character of Lesions

The simplest of all actinic injuries are sun burns which although frequently extensive with regard to percentage of body surface area involved are usually either first or second degree in depth Treatment is identical with that of thermal injuries by direct burn

Radiation injuries, in general are charac

terized by the fact that their ultimate gravity exceeds their initial apparent severity by the incompleteness of their healing (indolence) and by the late complications which ensue.

Pathology

Pathologic changes are induced not alone in the integument but in all tissues penetrated by the actinic rays. These include not only atrophy of skin but sclerosis of collagen in the depths of the wound and most important changes in the elastic lamina of blood vessels. The multiplicity of changes results in loss of function of appendages and deep viscera, and the hypertrophic changes in the skin at the edges of the ulcers produce keratoses and ultimately cutaneous carcinoma.

Complications

A first degree radiation burn produces an erythema of the skin in anywhere from 5 to 14 days. This type of burn ultimately will be depilatory, produce atrophic changes in the skin and be responsible for the late development of cutaneous telangiectasia (radiodermatitis). Second degree radiation injury produces an intense erythema after 5 or more days, edema of the skin and subcutaneous tissues and ultimately exfoliation and patent ulceration. The late sequelae of such burns are cicatrization (the formation of scar), hyperpigmentation, depilation and hyperkeratoses. Third degree radiation injuries produce excruciating pain because of involvement of nerves. A third degree necrosis of the skin is produced similar to a third degree thermal burn. Indolence in tractability to therapy of such lesions is due to damage to blood vessels in the depths of the wound. In the late postinjury period cutaneous carcinoma in the hyperkeratotic lesions is common. A chronic radiation dermatitis may include a combination of all of the above listed findings. Carcinoma of the skin develops in about 25 per cent of all cases of second and third degree actinic injuries.

Treatment

First and second degree actinic injuries are treated in a manner similar to treatment of thermal burns. Third degree actinic injuries are treated by wide excision of the complete thickness of the skin and ulcers and by grafting as soon as clinically feasible.

Radiation carcinogenesis demands much thought and analysis of clinical records as well as experimental investigation. It will continue to be a matter of great concern for organized medicine as well as government, because of its ever increasing utilization in medical therapeutics (particularly in intracavitary use and in therapy of thyroid disease) and because of the potential dangers in radioactive fallout following atomic tests.

SUMMARY OF PRINCIPLES

Burns

1. The successful management of extensive burns is a twofold problem: (*a*) the management of a patient with a profoundly disturbed physiology and (*b*) the problem of a huge open wound of a special type.

2. Burns are classified according to the depth of the wound: first, second, or third degree, and by the extent of the surface area involved.

3. The pathologic physiology of the local wound is an outpouring of plasma into the burned area and onto the surface of the wound owing to a profound vasodilatation and increased capillary permeability.

4. Edema fluid and burn fluid are essentially plasma.

5. The systemic pathologic physiology in extensive burns consists, se

quentially of (*a*) a diminished circulating volume (*b*) hemoconcentration, (*c*) diminished cardiac output, (*d*) tissue hypoxia (*e*) metabolic acidosis, (*f*) electrolyte imbalance and (*g*) hypoproteinemia.

6 Death from extensive burns from the time of the occurrence of the burn through the first 3 days is usually due to shock death due to anuria may follow the period of shock. Death from infection usually occurs between the fourth and seventh days Toxemic deaths usually occur from the seventh to the tenth days Death due to anemia usually occurs in the second week or later

7 Burn wounds are treated by gentle cleansing sharp debridement, and compression dressings where applicable In some areas and under some conditions the open local treatment may be employed Tetanus and gas gangrene must be guarded against, as in any large open wound

8 The local use of escharotic dyes and of antibiotic and chemotherapeutic ointments is not recommended

9 Burn wounds must be closed by suitable covering as early as possible in order to avoid persistence of the pathways for infection, toxemia disturbed nutrition and electrolyte imbalance

10 Partial thickness autologous skin grafts are the most desirable cover for burned surfaces

11 Homologous grafts may be of temporary value and may be lifesaving

12 The common infecting agents in burn wounds are the ubiquitous and sometimes remarkably resistant staphylococci, streptococci and colon paracolon bacilli *Ps aeruginosa* (*pyocyaneus*) is a frequent and troublesome contaminant.

13 The urinary bladder and upper urinary system and the respiratory tract are sites of common intercurrent infections

14 A complication which must be zealously guarded against is the occurrence of contractures as a result of excessive scar formation Careful resurfacing of flexion creases and immobilization in extension will guard against this undesirable end result

15 Management of the burn patient does not cease until rehabilitation to his prior usefulness is achieved Every plastic procedure and technic of physical medicine must be used to accomplish this desired result

Cold

1 Injury by cold may be due to actual freezing of the part (frostbite) actual cellular death occurs.

2 Immersion hand or immersion foot results from long exposure to low temperatures (above freezing) in the presence of moisture or high humidity

3 The parts most commonly involved in thermal injuries by cold are those parts which have a large surface area compared to total volume particularly the hands and fingers the feet and toes and the nose and ears

4 The pathologic physiology comprises (*a*) direct damage to cells in the case of actual freezing (as in burns) (*b*) the production of intra

vascular thrombosis by deposition of fibrin and sludging of cells in the case of immersion foot and (c) circulatory impairment due to pressure of the inflammatory exudate in both cases

5 Secondary infection may be superimposed on either type of damage

6 In the treatment of injury by cold thawing at room temperature minimizes tissue destruction

7 Anticoagulants are useful to prevent the sludging of cells and fibrin deposit which lead to intravascular thrombosis

8 Reflex vasodilatation by heat to other parts of the body by drugs and by sympathetic blocks are useful

9 Infection should be anticipated and treated

10 Elevation of the limb will promote venous and lymphatic drainage

Chemical Electric and Actinic (Radiation) Injuries

1 The treatment of chemical integumentary injuries depends on a knowledge of the material causing the burn and of its preferred neutralizing agents diluents or substances which cause it to be precipitated into an inactive form

2 Electric burns always result in an ulcer larger than would be expected from the initial evaluation This is due to a disintegration of the arterial media

3 Late complications of electric burns are hemorrhage and secondary infection

4 Electrocution is due to either ventricular fibrillation of the heart or respiratory center paralysis

5 Actinic injuries have a latent period affect all tissues that are penetrated by the rays and may destroy a part or the full thickness of the skin

6 Diffuse changes in the skin in actinic injuries involves sclerosis of collagen changes in elastic lamina of blood vessels and late hyperkeratotic lesions

7 About 25 per cent of all second degree and third degree actinic injuries result in carcinoma of the skin at the site of the wound

8 Third degree actinic injuries of skin must be excised widely and treated by grafting as soon as feasible

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7

Fractures and Amputations

I Fractures
  A Classification (1) nomenclature with respect *to skin (2) nomenclature* with respect to the bone (3) nomenclature with respect to associated injuries
  B Etiology (1) violence (2) disease
  C Pathologic physiology and mechanism of repair (1) organization within the hematoma (2) fibroplasia and granulation (3) formation of callus and osteoid (4) realignment of bone
  D Diagnosis
  E Methods of treatment
  F Complications (1) local (2) systemic
II Amputations
  A Classification
  B Indications
  C Types
  D Sites of election
  E. Rehabilitation of the patient

FRACTURES

Classification

A fracture is an interruption in or solution of continuity in the bone

The most important basis *for* classification of fractures is the presence or absence of an associated cutaneous wound A *closed* or *simple fracture* is one in which there is no associated wound through the overlying skin a *compound* or *open fracture* is one in which there is a communication between the site of the fracture and the outside of the body usually through a rent in the skin made either by external forces or by the end of one of the bone fragments. The significance of an open fracture lies in the fact that a broad avenue of contamination has been opened with all the potential sequelae of infection of deep tissues, including the medullary cavity of the bone

A *fracture* whether open or closed may be *complete* or *incomplete* Complete fractures may be either *transverse oblique* or *spiral* according to the mechanism of production An incomplete fracture may be incurred in a long bone particularly in children or in the ribs at any age A linear fracture in the skull or in the pelvic bones is often incomplete A *comminuted* fracture is one in which there are three or more fragments An *impacted* fracture is one in which two principal fragments are driven into each other resulting in fixation A *compression* fracture is one in which there is reduction in stature this applies particularly to the vertebral bodies.

Fractures may further be classified ac

cording to the relationship of the fracture line to the component parts of a long bone thus, the fracture may be of the *metaphysis* (the center of the shaft) of the *epiphysis* (the growing end of the bone) or *articular* (extending into an adjacent joint) A fracture may be complicated by associated soft tissue injury in the area of the fracture soft tissue injury refers particularly to muscles, blood vessels and nerves

Etiology

The causative agents in the production of fractures are (1) trauma and (2) disease Trauma may be applied in many ways (1) direct violence the application of force directly against a bone (2) indirect violence, usually caused by unusual muscular effort, such as twisting or turning (3) penetrating injuries such as gunshot wounds Fractures due to the weakening of bone structure by disease (*pathologic fractures*) occur with little or no precipitating trauma The underlying pathology is usually (1) neoplastic, primary or metastatic (2) congenital, i e defects in bone formation (3) endocrine such as the demineralization of hyperparathyroidism (4) atrophy of disuse, as is seen in certain neurologic disorders and after prolonged immobilization (5) infection chronic osteomyelitis

Pathologic Physiology and Mechanism of Repair

A fracture is an injury of a special type. The process of repair is essentially the same as in soft tissue wounds however there are some specific additional mechanisms which are adapted to the healing of this tissue Immediately after occurrence of the fracture the bone ends are surrounded by a hematoma and an inflammatory exudate which includes blood cellular elements and fibrin The reaction of this medium is acid in character under these conditions with the aid of osteoclasts spicules and rough edges of bone are decalcified and thus smoothed Some days later the character of the milieu becomes alkaline and osteoblasts become active permitting calcium to be deposited in the hematoma which is now organizing and which has become active granulation tissue This deposition of calcium together with the proliferation of fibroblasts (fibroplasia) from the periosteum and endosteum results in the formation of a matrix which is called *osteoid tissue* Chondroid tissue at the end of long bones undergoes similar changes without calcium deposition By the end of the second week of repair trabeculae appear in the osteoid tissue this entire area of reorganization around the fractured ends is called *callus* The amount of callus formed varies with the nature of the bone, being greatest in weight-bearing bones and least in bones that bear none As healing progresses, there is further deposition of minerals along lines of stress and absorption of rough ends and superfluous bone, while the medullary cavity of the bone is being restored. This is the process of *reossification* This entire course of healing of a fractured bone proceeds smoothly and efficiently to maximal reconstitution provided both local and systemic factors are optimal *Local factors* which are conducive to a favorable end result are (1) accurate apposition of fractured ends (2) no interposed tissue such as adjacent muscle (3) absence of an excessive amount of necrotic tissue (4) no infection (5) immobilization and (6) adequate blood supply *Systemic factors* which lead to good healing are (1) good nutrition, including normal hemoglobin and total protein levels and adequate vitamin supply particularly C and (2) absence of debilitating neoplastic, or metabolic diseases

Diagnosis

Fracture must be suspected whenever the body has been subjected to sufficiently severe trauma or when there is known or suspected pre-existing bone disease Pain is usually severe The signs of fracture are (1) pain (2) local tenderness, swelling and discoloration (3) loss of function in varying degrees (4) deformity such as shortening of an extremity, unusual appearance of

the limb or peculiar angulation (5) abnormal point of mobility along the course of the bone and (6) crepitus the sound of the bone ends grating one upon the other The examination should be made without inflicting additional pain and without aggravating deformity Manipulation should be kept to a minimum until definitive roentgenologic studies have been made in order not to contribute to shock and increase soft tissue damage

Methods of Treatment

The treatment of a patient with a fracture begins at the geographic site where the fracture was sustained Properly administered first aid and transportation to the hospital can prevent further injury and complications improper first aid and transportation can compound the injury with ultimate loss to the patient both in body function and in period of disability "Splint em where they lie" is a time honored idiom Any suitable splint which will immobilize a fractured limb will be conservative of time and function these need not be as well designed as the extremely useful Thomas splint, but may be boards or sticks or a house door Hemostasis for massive bleeding and the initiation of the treatment of shock take precedence over definitive therapy of the fracture they are discussed elsewhere

Reduction of the fracture to or close to normal should be accomplished as early as possible other serious injuries permitting. In closed fractures it is an accepted principle that, if accurate reduction can be accomplished by manipulation and external immobilization this is preferable to open operation An exception to this is the elective pinning of fractures of the femur to permit earlier ambulation Closed reduction can be accomplished by gentle manipulation of fragments, by the use of traction to bring the fragments into proper realignment, and by external methods to produce fixation and immobilization. Manipulation is always done gently and controlled by fluoroscopic or roentgenographic examination Fixation and immobilization by external means usually involve the application of a plaster cast or other (metallic) appliances When immobilization is effected one should always attempt to fix adjacent joints (wherever such inclusion is necessary) in the position of most useful function Traction on limbs is used for purposes of bringing the fragmented ends into alignment so as to make local factors conducive to healing Traction may be accomplished in one of two ways (1) skin traction by the application of a strong cloth cemented to the skin (2) skeletal traction via the insertion of a metal pin at a site distal to the fracture

The open or operative method of treatment of fractures is occasionally necessary in the treatment of closed fractures in order to obtain an optimal result The operative therapy of compound or open fractures is almost always mandatory exceptions are penetrating or perforating injuries with tiny skin wounds and no skeletal deformity The treatment of a compound fracture involves the basic principles of the therapy of wounds in addition to those which apply to the treatment of the broken bone After shock and hemorrhage have been properly controlled a formal operative attack must be made under the most suitable conditions in a regular operating room for the definitive treatment of a compound fracture This includes (1) continued support against incipient shock and anticipated blood loss (2) general anesthesia to eliminate pain and muscle spasm (3) cleansing of the wound (4) debridement including the removal of devitalized and necrotic soft tissue as well as detached fragments and extraneous dirt (5) the apposition of the fractured ends by the method which will produce and maintain the reduction by the simplest technic this may simply involve the application of external fixation by a plaster cast after meticulous closure of the wound or it may require internal fixation of one or another type Internal fixation may be accomplished by various types of hardware such as screws and/or plates applied through the fracture

cording to the relationship of the fracture line to the component parts of a long bone thus the fracture may be of the *metaphysis* (the center of the shaft) of the *epiphysis* (the growing end of the bone) or *articular* (extending into an adjacent joint) A fracture may be complicated by associated soft tissue injury in the area of the fracture soft tissue injury refers particularly to muscles, blood vessels and nerves

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The causative agents in the production of fractures are (1) trauma and (2) disease Trauma may be applied in many ways (1) direct violence the application of force directly against a bone (2) indirect violence, usually caused by unusual muscular effort, such as twisting or turning (3) penetrating injuries such as gunshot wounds. Fractures due to the weakening of bone structure by disease (*pathologic fractures*) occur with little or no precipitating trauma. The underlying pathology is usually (1) neoplastic, primary or metastatic (2) congenital i e., defects in bone formation (3) endocrine such as the demineralization of hyperparathyroidism (4) atrophy of disuse, as is seen in certain neurologic disorders and after prolonged immobilization (5) infection chronic osteomyelitis

Pathologic Physiology and Mechanism of Repair

A fracture is an injury of a special type The process of repair is essentially the same as in soft tissue wounds however there are some specific additional mechanisms which are adapted to the healing of this tissue Immediately after occurrence of the fracture the bone ends are surrounded by a hematoma and an inflammatory exudate which includes blood cellular elements and fibrin The reaction of this medium is acid in character under these conditions with the aid of osteoclasts spicules and rough edges of bone are decalcified and thus smoothed Some days later the character of the milieu becomes alkaline and osteoblasts become active permitting calcium to be deposited in the hematoma which is now organizing and which has become active granulation tissue This deposition of calcium together with the proliferation of fibroblasts (fibroplasia) from the periosteum and endosteum results in the formation of a matrix which is called *osteoid tissue* Chondroid tissue at the end of long bones undergoes similar changes without calcium deposition By the end of the second week of repair trabeculae appear in the osteoid tissue this entire area of reorganization around the fractured ends is called *callus* The amount of callus formed varies with the nature of the bone, being greatest in weight-bearing bones and least in bones that bear none As healing progresses, there is further deposition of minerals along lines of stress and absorption of rough ends and superfluous bone while the medullary cavity of the bone is being restored This is the process of *reossification.* This entire course of healing of a fractured bone proceeds smoothly and efficiently to maximal reconstitution provided both local and systemic factors are optimal *Local factors* which are conducive to a favorable end result are (1) accurate apposition of fractured ends (2) no interposed tissue, such as adjacent muscle (3) absence of an excessive amount of necrotic tissue (4) no infection (5) immobilization and (6) adequate blood supply *Systemic factors* which lead to good healing are (1) good nutrition including normal hemoglobin and total protein levels and adequate vitamin supply particularly C and (2) absence of debilitating neoplastic, or metabolic diseases.

Diagnosis

Fracture must be suspected whenever the body has been subjected to sufficiently severe trauma or when there is known or suspected pre-existing bone disease Pain is usually severe The signs of fracture are (1) pain (2) local tenderness swelling and discoloration (3) loss of function in varying degrees (4) deformity such as shortening of an extremity unusual appearance of

the limb or peculiar angulation, (5) abnormal point of mobility along the course of the bone and (6) crepitus the sound of the bone ends grating one upon the other The examination should be made without inflicting additional pain and without aggravating deformity Manipulation should be kept to a minimum until definitive roentgenologic studies have been made in order not to contribute to shock and increase soft tissue damage.

Methods of Treatment

The treatment of a patient with a fracture begins at the geographic site where the fracture was sustained *Properly administered* first aid and transportation to the hospital can prevent further injury and complications improper first aid and transportation can compound the injury, with ultimate loss to the patient, both in body function and in period of disability "Splint em where they lie" is a time-honored idiom Any suitable splint which will immobilize a fractured limb will be conservative of time and function these need not be as well designed as the extremely useful Thomas splint, but may be boards or sticks or a house door Hemostasis for massive bleeding and the initiation of the treatment of shock take precedence over definitive therapy of the fracture they are discussed elsewhere

Reduction of the fracture to or close to normal should be accomplished as early as possible other serious injuries permitting. In closed fractures it is an accepted principle that, if accurate reduction can be accomplished by manipulation and external immobilization this is preferable to open operation An exception to this is the elective pinning of fractures of the femur to permit earlier ambulation Closed reduction can be accomplished by gentle manipulation of fragments, by the use of traction to bring the fragments into proper realignment, and by external methods to produce fixation and immobilization Manipulation is always done gently and controlled by fluoroscopic or roentgenographic examination. Fixation and immobilization by external means usually involve the application of a plaster cast or other (metallic) appliances When immobilization is effected one should always attempt to fix adjacent joints (wherever such inclusion is necessary) in the position of most useful function Traction on limbs is used for purposes of bringing the fragmented ends into alignment so as to make local factors conducive to healing Traction may be accomplished in one of two ways (1) skin traction by the application of a strong cloth cemented to the skin (2) skeletal traction, via the insertion of a metal pin at a site distal to the fracture

*The open or operative, method of treat*ment of fractures is occasionally necessary in the treatment of closed fractures in order to obtain an optimal result. The operative therapy of compound or open fractures is almost always mandatory exceptions are penetrating or perforating injuries with tiny skin wounds and no skeletal deformity The treatment of a compound fracture involves the basic principles of the therapy of wounds in addition to those which apply to the treatment of the broken bone After shock and hemorrhage have been properly controlled, a formal operative attack must be made under the most suitable conditions in a regular operating room, for the definitive treatment of a compound fracture This includes (1) continued support against incipient shock and anticipated blood loss (2) general anesthesia to eliminate pain and muscle spasm (3) cleansing of the wound (4) debridement, including the removal of devitalized and necrotic soft tissue as well as detached fragments and extraneous dirt (5) the apposition of the fractured ends by the method which will produce and maintain the reduction by the simplest technic thus may simply involve the application of external fixation by a plaster cast after meticulous closure of the wound, or it may require internal fixation of one or another type Internal fixation may be accomplished by various types of hardware such as screws and/or plates, applied through the fracture

site or on the surface of the bone across the fracture site or a fixing nail used in the intramedullary cavity An excellent method of internal fixation is the use of bone grafts either free (from elsewhere in the body) or sliding (from areas adjacent to the fracture) In general autologous bone is preferable to metallic bodies which some times must be removed at a later date

The management of the patient with a fracture is not terminated until the patient is rehabilitated to or close to normal func tion Every means employed in physical medicine is used for the restoration of nor mal musculoskeletal function

Complications

The complications which may ensue fol lowing the treatment of fractures may be either local or general Local complications include those of poor healing (1) malunion in which healing takes place in such a way as to distort the function or appearance of a limb (2) nonunion in which the ends of a fractured long bone are united by fibrous tissue only and thus unable to bear weight (3) osteomyelitis the chronic infection of bone, discussed in detail elsewhere (4) in-juries produced by the fractured bone itself such as laceration of muscles, nerves and blood vessels. The general complications, resulting from long periods of immobiliza tion include (1) thromboembolic disease either thrombophlebitis or phlebothrombosis predisposed to by venous stasis aided and abetted by initial infection incurred at the time of injury (2) demineralization of the bones of the entire extremity from disuse (Sudeck s atrophy) (3) renal lithiasis, pre disposed to by the increased urinary ex cretion of calcium and rarely (4) fat em-bolism due to dislodgment of fat from the medullary contents into the venous system.

AMPUTATIONS

Classification

Amputation is the removal of all or part of a limb and includes disarticulations, in which the dismemberments are made through a joint. Amputations are classed according to the reason for which they are performed. These include (1) traumatic, resulting from external violence (2) pathologic or spon-taneous as from arteriosclerotic (dry) gan grene (3) purposeful which may be a mat ter either of election or of necessity

Indications

Broadly speaking, purposeful amputations are performed for the following reasons (1) to remove dead and devitalized tissue where all hope of repairing or restoring such a useless limb is gone (2) to preserve the patient s life when the limb harbors disease such as a tumor or an overwhelm ing infection which threatens the existence of the entire organism Specific indications include (1) the sequelae of trauma of all kinds (2) peripheral vascular disease, in-cluding arteriosclerosis obliterans thrombo-angiitis obliterans and arterial embolization and (3) malignancy The modern attitude with respect to amputation is to conserve all useful tissue and most important, function Such an attitude is permitted us in these times because of the restorative technics particularly of major arterial trunks and also those technics applicable to reconstitution of bone and nerve and plastic procedures for the "re-education" of muscles The practice of such *conservative surgery* in the true meaning of that term is made possible by the use of blood and blood substitutes, in addition to all other knowledge in the care of the body economy of the patient, the use of antibiotics and the availability of re frigeration anesthesia.

Types

Emergency amputations, of course are usually performed when the patient is either in shock or seriously ill from disease Under such conditions one does not prolong the time of anesthesia with a formal flap-forming amputation Rather one removes the dis eased or crushed limb as rapidly as possible

by the simplest method This the *guillotine amputation* has been practiced for centuries The amputation stump is allowed to close by granulation with or without traction on the skin to accomplish this end such a stump closure may be formally modified at a later time when the patient can better withstand more prolonged anesthesia Amputations performed as an elective procedure are designed to produce an optimal result from all points of view—function weight bearing and appearance—at the time of the initial operative attempt

Sites of Election

In general amputations should be done as far distally as is permitted by viable tissue However this principle is modified somewhat under two conditions if the amputation is done for malignant disease it must be done sufficiently proximal to the neoplasm to be reasonably sure that none remains the principle of amputation then bows to the principle of cancer surgery If the amputation is performed for occlusive arterial disease especially in the elderly patient it is sometimes preferable to perform a low thigh amputation when the adequacy of the circulation is questionable rather than subject these unfortunates to a second higher resection because of failure of healing. When amputation is performed for the removal of hopelessly traumatized tissue particularly in the young patient the rule of saving every bit of viable tissue is rigidly enforced in the upper extremity In the lower limbs certain sites of election are determined by the desirability and necessity of using prostheses thus, in a below the knee amputation the most desirable stump length for a properly functioning prosthesis is about 7 in below the knee joint

The multiplicity of functions performed by the upper extremities makes mandatory the accomplishment of amputations with the thought to the future use of every known device for the rehabilitation of the patient to economic usefulness and personal care Particular attention is paid to the restoration of functions of the hand and the digits In the lower extremity amputations through the lower third of the thigh must be suitable for weight bearing except in the very elderly By whatever technic accomplished such an amputation stump must be covered by a sufficient thickness of soft tissue to prevent decubitus of the skin produced by trauma to it between the end of the femur and the prosthetic appliance Also the sciatic nerve must be severed at a sufficiently high level so that it is not incorporated in the stump closure Below the knee amputations are most successfully fitted to a leg prosthesis if the tibia fragment is 7 in long. It is doubtful whether the placing of the patella over the cut end of the femur contributes anything to the supracondylar amputation from the point of view of making weight bearing easier Amputation of the foot leaving the heel bearing limb should not be used in the elderly or the patient with sclerotic vessels. The transmetatarsal amputation removing the distal halves of the metatarsals and all the toes is a very useful operation, particularly for the young diabetic who has occlusion of the digital vessels to the toes (usually with resulting gangrene osteomyelitis and cellulitis), it results in a useful foot with a normal gait

Rehabilitation of the Patient

Postoperative care is designed to avoid undesirable sequelae of amputations Position of maximal function of the amputation stump and the joint proximal to it must be maintained disabling flexion contractures are thus avoided. All modalities in physical medicine are employed for this purpose and for the maximal rehabilitation of the patient. Thromboembolic disease is avoided as much as possible by the use of anticoagulants prophylactic ligation of defunctionalized veins (such as the superficial femoral vein in a supracondylar amputation) is indicated.

SUMMARY OF PRINCIPLES

1 Fractures are classified with respect to the integrity of the overlying skin (closed or open) with respect to the bone (complete or incomplete transverse spiral or oblique) and with respect to the degree of complicating associated injuries (muscles nerves, and blood vessels)

2 Main causes of fractures are trauma and disease (pathologic)

3 A fracture is a special type of injury and is repaired through the usual mechanisms of fibroplasia and granulation tissue with the additional specific adaption mechanisms for the formation of callus osteoid and new bone

4 The diagnosis of fractures *is made by characteristic local findings* of pain tenderness, crepitus and loss of function and by x ray examination

5 The treatment of fractures begins with properly administered first aid and includes transportation to the hospital with the fracture immobilized

6 Reduction of fractures should be accomplished by the simplest possible method *preferably by a closed manipulative technic with subsequent* external immobilization. Suitable traction technics are frequently applicable

7 Open fractures are treated as any other complicated open wound after hemorrhage and shock have been properly controlled by a formal operative attack as detailed in Chapter 3

8 The complications following fractures may be either local (mal union nonunion osteomyelitis and associated visceral injuries) or systemic (thromboembolic disease, osseous demineralization and renal lithiasis)

9 Amputations may be accidental purposeful or pathologic

10 Purposeful amputations are performed (*a*) to remove hopelessly devitalized tissue and (*b*) for the preservation of the patient s life, if it is threatened by overwhelming infection or malignant disease in the limb

11 In all amputations the proper attitude is one of conservation of all useful tissue and most important of conservation of function

12 Reconstructive surgery should be used extensively for the preservation of function of the forearm hand and digits.

13 Every modality in physical medicine should be employed for the rehabilitation of the patient to economic and personal usefulness.

SUGGESTED READINGS

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8

Craniocerebral Injuries

- I Introduction
- II Scalp
- III Skull
- IV Brain damage
  - *A* Anatomy and physiology
  - *B* Types and mechanisms (1) concussion (2) contusion (*a*) direct (*b*) *contrecoup* (3) hemorrhage (with compression) (*a*) intra cerebral (*b*) epidural (*c*) subdural acute and chronic
  - *C* Pathologic physiology
- V Examination of the patient
  - *A* Vital signs (1) level of consciousness (*a*) confusion (*b*) semicoma (*c*) coma (2) blood pressure, pulse, temperature, respiration
  - *B* Local external findings
  - *C* Pupils
  - *D* Fundi
  - *E* Motor power
  - *F* Reflexes
  - *G* Skull x-ray examination
  - *H* Lumbar puncture
  - *I* Associated injuries
- VI Treatment
  - *A* General
  - *B* Operative (1) depressed fractures (2) epidural hemorrhage (3) subdural hemorrhage

INTRODUCTION

Head injuries are caused by many modalities in a heavily populated and highly industrialized society such as ours. The automobile age has brought an appalling increase in the numbers of persons maimed or killed in traffic accidents Highway casualties have mounted to a position high in the list of causes of death in which they have supplanted tuberculosis and pneumonia This slaughter must be halted by legal and technologic means Nonetheless, there is every expectancy that accidents and, with them injuries to the head will continue to constitute a major surgical problem. The initial evaluation and care of the patient with a craniocerebral injury, usually by the intern on call in the emergency room or by the doctor in general practice who is first summoned, may determine the issue of survival or death At best, because of the very special surgical anatomy of the brain and of the physiology of its vital tissues, the therapy of

these serious injuries leaves much to be desired. A basic understanding of the principles which follow from a knowledge of neurophysiology and its disturbance subsequent to trauma to the head is a *sine qua non* for the maximal salvage of life Fewer errors of omission and of commission will be made if management is carried out, not by rote but according to reasoning based on these fundamentals

SCALP

The scalp from the outside to the outer table of the skull, consists of five layers (1) skin, (2) subcutaneous tissue, (3) galea aponeurotica, (4) loose areolar tissue, and (5) periosteum

Injuries to the scalp are of several types (1) contusion, (2) hematoma, (3) lacerations Hematomas of the scalp are of several types and vary with their location within the layers of the scalp A subcutaneous hematoma has an area of central softness if the *hematoma is subgaleal, it will be diffuse and* may be very extensive If bleeding is subperiosteal the hematoma will be sharply demarcated by the geographic limitations of the sutures of the skull In any case the hematoma may be so great that it can be depressed centrally by the finger not uncommonly leading to the false impression of a depressed fracture Lacerations of the scalp are caused by many types of trauma this is because of the nature of scalp injuries—the crushing of a thin bit of soft tissue between two hard objects, the one delivering the blow the second the outer table of the skull Bleeding from scalp lacerations is vigorous because of the abundant blood supply By the same token it is very difficult to devascularize the scalp and therefore healing is usually rapid and complete Hemostasis for apparent massive bleeding of the scalp can easily be obtained temporarily by digital pressure or by applying hemostats to the *galea and turning them outward from* the lacerated wound This causes the blood vessels in the subcutaneous tissue and the loose areolar tissue on both sides of the galea to be turned upon themselves and temporarily compressed The usual treatment of a soft tissue wound can then be carried out at leisure this includes adequate cleansing after shaving of the hair around the wound, and debridement. Ligature of bleeding vessels can then meticulously and leisurely be accomplished and exploration for fractures of the skull can be made with preparations for elevation should it be deemed necessary Closure of the wound will follow those principles for closure of traumatic wounds anywhere discussed in Chapter 3

SKULL

There is a tendency even among physicians, to lay too much stress on the presence or absence of fractures of the bones of the skull which only occasionally are important. Much more significant is the degree of injury to the brain tissues There are various types of fractures of the skull the most common are *linear fractures* of the vault. Unless associated with the tear of a large epidural vessel these are usually of little importance A *depressed skull fracture* is one *in which a portion of the inner table* of the skull has been driven down (depressed) toward or through the dura mater and into cerebral tissue As in all fractures, those of the skull may be closed or open (compounded by an associated laceration of the scalp above it) Common sites of both linear and depressed fractures of the skull are the temporal parietal frontal and occipital areas The basilar portion of the skull may be fractured in any of its three fossae Fracture through the basal portion of the anterior fossa may be indicated by massive periorbital hemorrhage and by cerebrospinal fluid rhinorrhea middle fossa basilar fracture may be indicated by hemorrhage into the middle ear and by cerebrospinal fluid otorrhea if the tympanum is ruptured posterior fossa basilar fracture may be indicated by cerebellar symptoms. Unusual fractures are those produced by a bursting mechanism

in which the fractures radiate from the site of applied trauma and fracture by *contre coup* wherein the fracture occurs at a site in the vault 180° opposite to the site of applied trauma because of the focusing of lines of stress.

BRAIN DAMAGE

Anatomy and Physiology

The essential consideration in craniocerebral injuries is the damage to the contents of the cranial vault, the brain Cerebral edema may result directly from the injury or as a late sequel to changes initiated at the time of injury

Types and Mechanisms

There are three main types of brain damage *Concussion* is the immediate traumatic interruption of function which occurs in the absence of visible lesions in the nervous system There is loss of consciousness of short duration with respiratory and circulatory changes as well These may be followed by confusion headache and unsteadiness, which may endure for minutes, hours, or days According to this definition concussion is a diagnosis in retrospect. Examples of concussion are the knockout punch of boxing and the blackjack injury

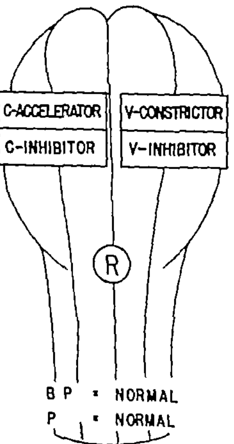
Contusion of the brain is always accompanied by edema and usually by hemorrhage and may be associated with gross laceration of brain tissue It may be direct, i e. directly beneath the area of skull that has been traumatized, or *contrecoup* i.e. at a site 180 from the location of the externally inflicted trauma. This is due to the vibration and bouncing of the brain against the opposite inner table, permitted by the slight mobility of the brain within the cranial vault. Thus signs of occipital contusion may be found when a blow is struck upon the forehead.

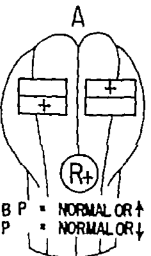
The third form of brain damage is due to actual *compression* of nervous tissue it may be due to (1) depressed bone fragment, (2) extradural compression (3) subdural compression or (4) intracerebral hemorrhage Far and away the most serious of all types of brain damage is that due to hemorrhage into nervous tissue Any part of the brain may be so severely contused as to result in hemorrhage within its substance Hemorrhage into the internal capsule can produce many bizarre neurologic symptoms whereas hemorrhage into the cerebral motor cortex may result in immediate crossed hemiplegia Even if death does not follow intracerebral hemorrhage severe disability usually results, because the organization of intracerebral hematomas results in gliosis of brain tissue with functional destruction

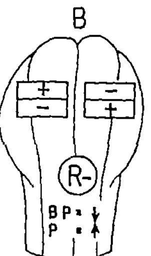
Compression of the brain by epidural (extradural) hemorrhage is usually the result of laceration of an arterial trunk lying between the dura and the inner table of the skull a gradual hemiplegia usually occurs in such cases Compression of the brain as a result of subdural hemorrhage is usually venous in nature and occurs when the subdural veins are sheared off Unlike intracerebral hemorrhage, epidural and subdural hemorrhage may be treated successfully

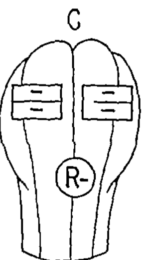
Pathologic Physiology

The pathologic physiology of cerebral injury will be traced from a minimal and reversible degree to the most extensive and irreversible changes (Fig. 11) The local damage to a small area of neural tissue promptly leads to edema in the affected and the adjacent area if minor there may be signs and symptoms referable to the area involved, and then subsidence of the edema will result in clinical recovery If the area of local destruction is greater the edema is correspondingly widespread and may be enough so to lead to a compression of the outflow tracts of cerebrospinal fluid and of venous blood from the cranial vault When the resulting *rise in intracranial pressure* is great enough to obtund the inflow of arterial blood into the cranial vault as it flows toward the brain, certain compensatory reflexes are initiated which elevate the systemic blood









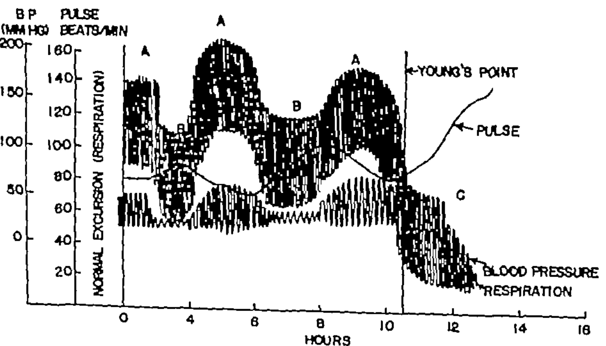


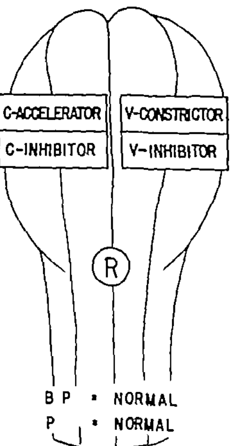
FIG. 11 Graphic representation of

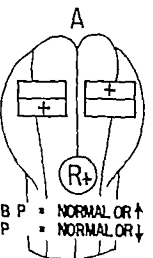
pressure This increased systolic blood pressure exceeds at first the intracranial pressure and permits the further entrance of arterial blood to the brain which again may be partially obstructed by continued rise in intracranial pressure Thus there will be an alternately interrupted and resumed arterial cerebral blood inflow The brain under these conditions is subjected to intermittent sufficiency and insufficiency of oxygen which adversely affects the various *vital centers* particularly in the medulla and this intermittency is responsible for the vacillations in the vital signs found in the patient with a head injury

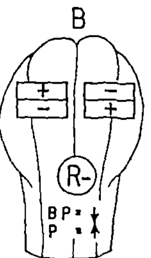
The regions most seriously affected are the (1) *respiratory* (2) *vasomotor* and (3) *cardiac centers* The cardiac center of the medulla consists of two portions which can be independently stimulated, the *cardioaccelerator* and the *cardioinhibitor divisions* This is likewise true of the vasomotor center consisting of the *vasoconstrictor* and the *vasodilator portions* Hypoxia initially results in stimulation of nervous centers but as it becomes more severe these centers become exhausted The initial hypoxia causes the following sequence to take place First the cardioinhibitor portion of the cardiac center and the vasoconstrictor portion of the vasomotor center become stimulated This usually results in the early findings of a relatively normal or slightly elevated systolic systemic blood pressure with a normal heart rate or even a slight bradycardia. With increasing intracranial pressure and cerebral hypoxia these divisions are depressed and the somewhat less sensitive cardioaccelerator and vasodepressor centers are now stimulated, with a resulting drop in systolic blood pressure and a rise in heart rate Compensatory mechanisms are then brought into play which elevate systemic arterial pressure and the process reverts to its original status in which the cardioinhibitor and vasoconstrictor centers are again stimulated The compensatory reflexes referred to are those initiated by the pressure receptors and chemoreceptors in the arch of the aorta and in the carotid sinuses The afferent components of the reflexes excited either by changes in pressure or by blood content of oxygen and carbon dioxide stimulate or depress the medullary target centers resulting in peripheral vasomotor changes The alternate stimulation and therefore dominance and exhaustion and therefore inactivation of the various centers is graphically depicted in the diagram in Figure 11 Should the rising intracranial pressure continue unabated ultimately the vasoinhibitor center and the cardioaccelerator center are maximally stimulated with a precipitous drop in systemic systolic blood pressure and a marked tachycardia When the heart rate in beats per minute exceeds the systolic blood pressure in millimeters of mercury and they both continue in that direction Young s point has been reached and crossed a critical state

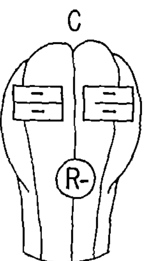
During this time when the vasomotor and cardiac centers have been struggling to maintain an adequate cerebral oxygen supply and fluctuations in the degree of deficiency have been reflected in the vacillating vital statistics, another center has been severely disturbed the *respiratory center* The first impact of hypoxia upon the respiratory center produces stimulation there is an increased respiratory rate (*tachypnea*) and an increased amplitude of each inspiratory movement. As hypoxia progresses the respiratory center becomes exhausted this is indicated by a diminished rate (*bradypnea*) as well as by a shallow excursion When the compensatory arterial mechanisms come into play bringing more blood to the brain and the hypoxia of the respiratory center is diminished it again is stimulated and the respiratory excursion and rate are again increased this alternating hypoxia and improved oxygenation of the respiratory center reflects itself in periods of relative *apnea* and of relative respiratory sufficiency the phenomenon of *Cheyne Stokes respiration* Reference to the chart (Fig. 11) will clarify the pathologic physiology of raised intracranial pressure

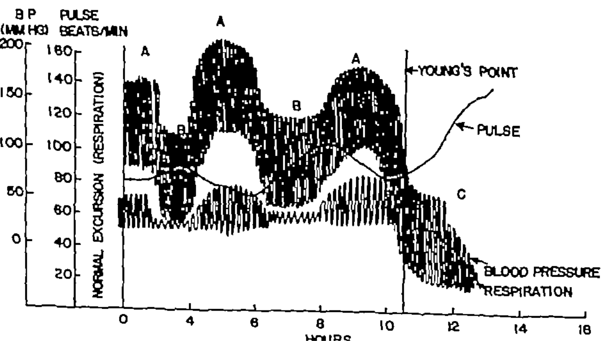
The pathologic physiology of increased in


NORMAL
C-ACCELERATOR
C-INHIBITOR
V-CONSTRICTOR
V-INHIBITOR
R
B P = NORMAL
P = NORMAL


A
+
+
R+
B P = NORMAL OR ↑
P = NORMAL OR ↓


B
+
−
−
+
R-
B P = ↓
P = ↑


C
−
−
−
−
R-

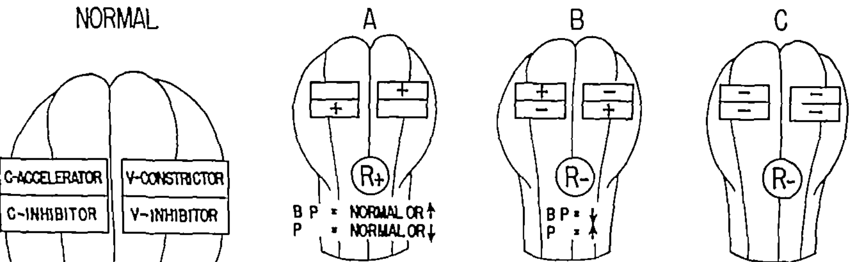

B P (MM HG)
PULSE BEATS/MIN
200
150
100
50
0
160
140
120
100
80
60
40
20
NORMAL EXCURSION (RESPIRATION)
A
B
A
B
A
C
YOUNG'S POINT
PULSE
BLOOD PRESSURE
RESPIRATION
0
4
6
8
10
12
14
16

pressure This increased systolic blood pressure exceeds at first the intracranial pressure and permits the further entrance of arterial blood to the brain which again may be partially obstructed by continued rise in intracranial pressure Thus there will be an alternately interrupted and resumed arterial cerebral blood inflow The brain under these conditions is subjected to intermittent sufficiency and insufficiency of oxygen which adversely affects the various *vital centers* particularly in the medulla and this intermittency is responsible for the vacillations in the vital signs found in the patient with a head injury

The regions most seriously affected are the (1) *respiratory* (2) *vasomotor* and (3) *cardiac centers* The cardiac center of the medulla consists of two portions which can be independently stimulated the *cardioaccelerator* and the *cardioinhibitor divisions* This is likewise true of the vasomotor center consisting of the *vasoconstrictor* and the *vasodilator portions* Hypoxia initially results in stimulation of nervous centers but as it becomes more severe these centers become exhausted The initial hypoxia causes the following sequence to take place First the cardioinhibitor portion of the cardiac center and the vasoconstrictor portion of the vasomotor center become stimulated This usually results in the early findings of a relatively normal or slightly elevated systolic systemic blood pressure with a normal heart rate or even a slight bradycardia With increasing intracranial pressure and cerebral hypoxia these divisions are depressed and the somewhat less sensitive cardioaccelerator and vasodepressor centers are now stimulated with a resulting drop in systolic blood pressure and a rise in heart rate Compensatory mechanisms are then brought into play which elevate systemic arterial pressure and the process reverts to its original status in which the cardioinhibitor and vasoconstrictor centers are again stimulated The compensatory reflexes referred to are those initiated by the pressure receptors and chemoreceptors in the arch of the aorta and in the carotid sinuses The afferent components of the reflexes excited either by changes in pressure or by blood content of oxygen and carbon dioxide stimulate or depress the medullary target centers resulting in peripheral vasomotor changes The alternate stimulation and therefore dominance and exhaustion and therefore inactivation of the various centers is graphically depicted in the diagram in Figure 11 Should the rising intracranial pressure continue unabated ultimately the vasoinhibitor center and the cardioaccelerator center are maximally stimulated with a precipitous drop in systemic systolic blood pressure and a marked tachycardia When the heart rate in beats per minute exceeds the systolic blood pressure in millimeters of mercury and they both continue in that direction Young s point has been reached and crossed a critical state

During this time when the vasomotor and cardiac centers have been struggling to maintain an adequate cerebral oxygen supply and fluctuations in the degree of deficiency have been reflected in the vacillating vital statistics another center has been severely disturbed the *respiratory center* The first impact of hypoxia upon the respiratory center produces stimulation there is an increased respiratory rate (*tachypnea*) and an increased amplitude of each inspiratory movement As hypoxia progresses the respiratory center becomes exhausted this is indicated by a diminished rate (*bradypnea*) as well as by a shallow excursion When the compensatory arterial mechanisms come into play bringing more blood to the brain and the hypoxia of the respiratory center is diminished it again is stimulated and the respiratory excursion and rate are again increased this alternating hypoxia and improved oxygenation of the respiratory center reflects itself in periods of relative *apnea* and of relative respiratory sufficiency the phenomenon of *Cheyne-Stokes respiration* Reference to the chart (Fig 11) will clarify the pathologic physiology of raised intracranial pressure

The pathologic physiology of increased in



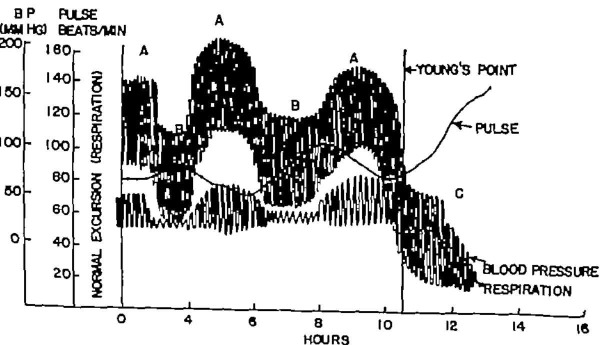


FIG. 11 Graphic representation of pathologic physiology of raised intracranial pressure See text for explanation

complished by ligation of the bleeding artery in its groove on the inner side of the inner table of the skull

Fundi

Funduscopic examination is of great value in detecting and following the course of elevated intracranial pressure The findings may vary from mere venous engorgement of the fundi to retinal hemorrhages later a blurring of the disk margins may be detected ultimately with obliteration to the point of producing a *choked disk* which following trauma is synonymous with raised intracranial pressure

Motor Power

An assessment of *motor power* even in an unconscious patient is of value Motion of the extremities in response to painful stimulus is characteristic of a semicomatose state this is a step lower in the state of consciousness than when there is spontaneous muscular activity Even when there is no muscular response to stimuli degrees of *tonicity* of the muscles can be demonstrated By elevating the lower extremity for several inches and dropping it suddenly some reflex tonicity of muscle is indicated if the extremity falls not precipitously but in stages when the extremity falls as dead weight with no evidence of tonicity of muscles, the patient is indeed deeply comatose In the comatose patient occasionally a facial nerve paresis may be an indication that a hemiplegia exists.

Reflexes

In the recording of *reflexes* both deep (tendon) and superficial (skin) the differences between the two sides and their variation with time are most important in assessing the progress of this pathologic state

Skull X Ray Examination

Roentgen-ray examination of the skull is more often made too early than too late If a depressed fracture is suspected usually by palpation of a depression in the skull it is justifiable to take the patient to the x ray room However roentgen ray examination of the skull can usually safely wait until therapy has been instituted for the correction of severe associated injuries and raised intracranial pressure In the presence of serious injuries the movement of the patient onto and from the x ray table and the time consumed thereby may be harmful and are therefore contraindicated

Lumbar Puncture

The subject of *spinal (lumbar) puncture* is deliberately placed last in this list of items in the examination Lumbar puncture immediately upon admission to the emergency room is of no practical value If elevated intracranial pressure already exists lumbar puncture is actually dangerous at this stage because rapid release of cerebrospinal fluid pressure through a hole in the lumbar dura will cause the brain to be herniated toward or through the foramen magnum with rapid death The imprint of the foramen magnum on the medulla the *conus medullaris* has been not uncommonly seen at autopsy following release of too much fluid via lumbar puncture Lumbar puncture may be employed after a day or two but then only for diagnostic purposes with a small caliber needle but not to withdraw fluid as a therapeutic measure The information desired the pressure of the fluid and whether blood is present or not can be gained with the first few drops of fluid released the lumbar puncture should be discontinued at that point.

Associated Injuries

The patient who has sustained a head injury has usually suffered *associated* injuries of other parts of the body Examination and treatment of the patient must, of course include all injuries In multiple injuries, a state of shock on admission to the emergency room is probably due to some injury other than that of the head Examination of the thorax for normal respiratory function the abdomen for evidence of massive bleeding

tracranial pressure is not manifested by the signs and symptoms referable to the medullary centers alone Irritation of the cerebrum, particularly by a state of hypoxia, results in the characteristic picture of *restlessness* This is the same type of restlessness occasionally seen in a patient under anesthesia, commonly misinterpreted as the result of too light a plane of anesthesia when really it is the first sign of cerebral underoxygenation Massive hemorrhage in and around the basal *areas of the brain including the medulla* and the hypothalamus causes severe disturbance *of vital functions that are usually irreversible* Under such conditions when the patient is first seen he is observed to be hyperventilating with both an increased amplitude of respiratory excursion and an increased respiratory rate (*hyperpnea*) this hyperventilation responds to no known clinical therapy Usually associated with such a finding is a *hyperpyrexia* which is usually present when the patient is admitted to the emergency room it is due to maximal stimulation of the heat regulatory center in or near the hypothalamus.

EXAMINATION OF THE PATIENT

Vital Signs

Changes in consciousness of varying degrees, are a characteristic manifestation of head injuries The assessment of conscious level must be made upon admission as a base line and at frequent intervals thereafter Variation in the state of consciousness is a general and important indication and criterion of the functional state of the brain *Confusion* is a disturbance of consciousness characterized by the inability to think clearly or to respond to a situation *Semicoma* is a deeper state in which the patient responds with motor movements to painful stimuli *Coma* is absolute unconsciousness with no response to any kind of stimuli Life is then on a reflex basis usually at the medullary and cord levels

The other vital signs, temperature pulse, respiration, and blood pressure must be followed from the time of admission at very frequent intervals Not only is the base line of these findings necessary but the changes in each one of them will help to indicate the progression of the pathologic physiology in and around the brain *These are most lucidly* recorded on the chart in a graphic manner

Local External Findings

The location of external evidences of violence aids in directing the attention to certain aberrations *of physiology* The presence, location, and extent of lacerations, contusions fluid, and blood leaks are all valuable and may be indicative of the major site of intracranial damage They may be a guide to thinking in the presence of direct or *contrecoup* contusions and may localize a basilar fracture.

Pupils

Examination of the *pupils* upon admission and at frequent intervals thereafter is important in assessing intracranial pathology and in following the patient s course The size reaction and equality *of* the pupils *must be observed and recorded, preferably* graphically Bilaterally fixed and dilated pupils *are an ominous sign they* are usually found in severe intracerebral hemorrhage. *Small contracted pupils (in the absence* of any administered narcotics) usually indicate severe *medullary damage* Pupillary inequality with impairment of the light reflex on the side of the unilaterally fixed, dilated pupil indicates one of the following conditions (1) extradural hemorrhage *which must* be assumed until specifically excluded (2) herniation through the tentorium (3) *torn* oculomotor nerve at its origin (4) injury to the oculomotor nerve in the orbit by bone spicules The importance of this set of findings cannot be overestimated, since they may indicate an extradural hemorrhage on the ipsilateral side a situation which is *remediable* by early operative intervention This is ac

loss of protein is almost inevitable without oral intake (7) *Oral hygiene* must be meticulous in order to prevent infection of the salivary glands, both parotid and submaxillary. Poor oral hygiene is conducive to the development of infections of the buccal mucous membrane by spirilla and fungi (8) An *indwelling catheter* must be kept in the bladder of the unconscious patient in order to avoid maceration of skin and decubitus ulceration (9) *Biotherapy* must be employed in order to avoid infection of the respiratory tree as well as of the urogenital tract particularly with the use of an indwelling catheter (10) *The patient must be turned frequently* in order to avoid decubitus ulcers of the sacrum and the bony prominences of the pelvis.

Operative

Operative therapy is required in less than 20 per cent of all cases of head injuries. It is regrettable that the neural tissue of the brain is not as amenable to treatment as are other soft tissues this factor is responsible for the high rate of morbidity and mortality The following conditions are amenable to operative therapy (1) *Depressed fractures* of the skull are logically submitted to operative intervention particularly if associated with an open wound (a compound fracture) Elevation of the depressed fragments not only relieves the existing compression but also prevents further edema and potential infection (2) Compression of the brain by *epidural (extradural) hemorrhage* is the result of laceration of an artery which lies between the inner table of the skull and dura This lesion produces a characteristic picture and timely operation may yield spectacular results. The typical history is that of an injury to the head perhaps associated with a transitory unconsciousness (concussion) followed by a lucid interval of minutes to hours after which there is a gradual lapse back into coma At this stage there is often evidence of a fixed dilated pupil on the side of injury with, sometimes contralateral hemiplegia Operative release of pressure and ligature of the bleeding vessel often reverses the state of consciousness on the operating table When a patient who has been unconscious and then has a period of relative lucidity appears sleepy or drowsy or unconscious it is imperative to determine which of these states exists is he sleepy or is he unconscious? Such a patient must be aroused every 2 hr during the first 24 hr this will determine whether this is a natural sleep or whether he is really unconscious If this is a second period of unconsciousness and the pupillary signs and symptoms clearly indicate an extradural hemorrhage operative intervention must be undertaken without delay If doubt exists and the patient is deteriorating rapidly one may make burr holes in the skull in an attempt to detect epidural bleeding The second episode of unconsciousness in epidural bleeding (arterial in nature) comes within 4 to 8 hr after trauma (3) *Subdural hemorrhage* from tearing of small veins between the meninges and the brain presents a very different clinical picture Subsequent to what may have been a mild and overlooked head injury there is a slow and progressive development of brain compression resembling that in brain tumor in which intractable headache is the predominant symptom which may eventuate in coma. This sequence extends over a period of hours to days or even weeks and the relationship to the head injury may be lost Early fluid blood may be spread over the brain surface In late cases the hemorrhage becomes organized and sometimes encapsulated but because of the greater osmotic tension in this hematoma, fluid is drawn into it thus explaining its slow but relentless growth The mechanism of production of unconsciousness whether by *epidural* or subdural accumulation of blood is *compression* of the brain The compression may in the extreme be bilateral owing to marked depression of the cortex under the hemorrhage and the pushing of the entire brain toward the opposite side with pressure against the rigid

or ruptured hollow viscera, and the extremities *for deformity and severe hemorrhage* must never be omitted Even though craniocerebral injuries take precedence over the treatment of less critical injuries the treatment of shock, massive hemorrhage and ruptured hollow viscera must be given priority The finding of these conditions may be masked by the cranial signs unless complete and meticulous examination is made

TREATMENT

General

In the therapy of a patient with a head injury it must be emphasized that one is treating not just a head injury but the whole patient who cannot now care for himself Therefore, the *general treatment* of a patient who has sustained a craniocerebral injury must be complete and detailed (1) An *unobstructed airway* must be obtained and maintained this is an absolute essential in the therapy of the unconscious patient. The airway may be maintained simply by permitting postural drainage with the patient on his side and the mouth pointing downward repeated frequent aspirations of the mouth and pharynx may be necessary to remove secretions which might otherwise obstruct the oropharyngeal tracheal airway It may be necessary to administer oxygen by any suitable route to compensate for ventilatory deficiency due to unremoved secretions *Tracheostomy* is frequently of great help in maintaining the airway and clearing tracheobronchial secretions in the unconscious patient the time to do the tracheostomy is when doubt exists whether one is necessary (2) *Shock* is not common in uncomplicated head injuries when shock is present associated injuries must be looked for in a systematic manner and treated according to *accepted principles detailed elsewhere* (3) *Hyperthermia* of hypothalamic damage must be treated by all suitable nursing technics reduction from hyperthermic levels will reduce oxygen requirements, make fluid and electrolyte control more successful and prevent exhaustion of certain body vitamins (particularly C) and enzymes. (4) The *restlessness* of cerebral hypoxia assuming that *oxygen is being administered and an open* airway is being maintained, is best treated with paraldehyde It is far safer and more effective than barbiturates narcotics are contraindicated lest respiratory depression ensue. Chloral hydrate is also a relatively safe drug (5) *Fluid and electrolyte balance* are no less important in the unconscious patient with a head injury than in any other patient. The withholding of fluids from such a patient for the reason that the brain will thus be dehydrated and shrunk is illogical and not supported by any evidence Cerebral edema is not dependent on normal body fluid requirements and the withholding of such maintenance requirements is not beneficial for the existing cerebral edema It is not illogical to maintain fluid and electrolyte balance as it is done elsewhere and at the same time, to use concentrated intravenous solutions in an attempt to shrink the brain. However there is a great deal of doubt on the part of neurosurgeons that these hypertonic solutions really shrink the brain for more than a few minutes and there is evidence that a reactive edema occurs which may be worse than the original The use of concentrated human albumin has been attended with more success than the use of concentrated crystalloids Of the new substances, lyophylized urea gives most promise of producing maximal cerebral shrinkage without reactive edema There is a growing body of evidence that hypothermia, induced in the patient with cerebral edema, is protective to the brain through the reduction of its oxygen requirement and this modality of therapy is becoming increasingly useful and popular (6) The unconscious patient who is maintained parenterally will lose weight unless *adequate calories and vitamins* are supplied It is difficult to maintain weight over a long period of unconsciousness since

calvarium (4) *Chronic cerebrospinal fluid rhinorrhea* indicating a fracture of the cribri form plate and an associated tear of the dura, usually subsides spontaneously occa sionally operative intervention is necessary to avoid ascending meningitis.

SUMMARY OF PRINCIPLES

1 The initial evaluation and care of the patient with a craniocerebral injury by the first doctor who is summoned may determine the issue of survival or death

2 A basic understanding of the principles which follow from a knowl edge of neurophysiology and its disturbance subsequent to trauma to the head is indispensable for the maximal salvage of life. Management must be carried out, not by rote but according to reasoning based on these funda mentals

3 The scalp consists of five layers of tissue including an abundant blood supply The presenting picture of hemorrhage in the scalp may vary greatly depending upon the location of extravasated blood in the layers of the scalp If the hematoma is subcutaneous, it may have a central soft spot if it is subgaleal it may be extremely extensive and diffuse if it is under the periosteum it will be limited by the sutures of the skull In any case depression of the hematoma by the examining finger may give the false im pression of a depressed skull fracture

4 In general too much stress is laid on the presence or absence of fractures of the bones of the skull Far more important is the question of injury to the underlying brain tissue Little need be done for linear fractures of the skull and not much is possible for basilar fractures of the skull

5 The underlying damage to the brain may be at the site of external violence or contralateral to it, at 180° from the signs of external violence

6 There are three main types of brain damage (1) concussion (2) contusion and (3) compression

7 Concussion is the immediate traumatic paralysis of cerebral func tion which occurs in the absence of visible lesions in the nervous system One makes the diagnosis of concussion therefore in retrospect. Contusion of the brain is always accompanied by a varying degree of edema and may be associated with hemorrhage

8 Compression of the brain may be caused by (1) a depressed frac ture or (2) hemorrhage outside the dura, underneath the meninges or into brain substance itself

9 The pathologic physiology of raised intracranial pressure is best understood when it is appreciated that neural tissue at first is stimulated by hypoxia and by compression but ultimately is exhausted by these same stimuli There is a vacillating oxygen sufficiency and insufficiency to the vital medullary centers controlling the muscles of respiration the heart rate and the tonus of peripheral blood vessels This alternating metabolic insufficiency accounts for the changing clinical picture of the patient with a craniocerebral injury The vital signs including temperature pulse heart

9

The Autonomic Nervous System

I General anatomic considerations: divisions and relation to central nervous system
II General functional considerations
   A Sensory and motor components
   B Concept of homeostasis
III Methods of study and of intervention
   A Pharmacologic
   B Surgical
IV Diseases
   A Sympathicotonia (1) Raynaud's phenomenon (2) thromboangiitis obliterans (3) trauma per se (4) causalgia (5) hypertension (6) other
   B Parasympathicotonia (vagotonia) (1) peptic disease (2) asthma (3) pancreatitis (4) ulcerative colitis
   C Diseases of the autonomic nerves themselves

GENERAL ANATOMIC CONSIDERATIONS

The autonomic nervous system has two distinct parts (1) the *sympathetic or thoracolumbar system* and (2) the *parasympathetic* or *craniosacral system* Each originates from cells in the central nervous system either in the brain stem or in the spinal cord The cells communicate with the viscera by way of two axones a preganglionic and a postganglionic, with an intervening ganglion In the case of the sympathetic nervous system the ganglia are located in a paravertebral chain which extends from the superior cervical ganglion superiorly to the last lumbar usually at the level of the fourth lumbar vertebra caudad The cells of the parasympathetic system have their synapsing ganglia in or near the viscera which they innervate

GENERAL FUNCTIONAL CONSIDERATIONS

Sensory and Motor Components

Within the autonomic nervous system there are visceral motor and sensory nerves somatic autonomic innervation is motor in nature there being no proof of somatic sensory fibers.

Concept of Homeostasis

The functions of the autonomic nervous system are *adaptive* in nature The sympathetic and parasympathetic divisions are antagonistic and reciprocal to each other where one stimulates, the other depresses when one is active the other is passive Together they constitute an important link in the maintenance of relative stability of both visceral and somatic functions by providing a rapid means of reacting to sudden changes

20 Compression of the brain due to subdural hematoma characteristically occurs at a later stage after the initial injury than does compression from an extradural hemorrhage. This is because it is venous in nature plus the fact that the subdural hematoma becomes larger with time, owing to its attraction of fluid because of its oncotic pressure Intractable headache due to chronic subdural hematoma may follow the initial trauma by such a long period of time that the relationship to the head injury may be lost.

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to ablation of the vagus nerve which is known to be one of the natural stimulants to the secretion of that organ Although the hormone *secretin* which is manufactured in the duodenum controls the concentration of the exocrine products in secretions of the pancreas, it is nevertheless true that stimulation of the vagus nerves produces a secretion of greater volume but of lower enzyme content Although only partially remedial in this serious situation parasympathicolytic drugs are nonetheless logical Surgical vagotomy is not indicated in the light of recent work on the pathogenesis of this disease

From time to time surgeons have suggested the operation of vagotomy for the control of the disease known as *nonspecific ulcerative colitis* For those who believe (without conclusive evidence) that ulcerative colitis is due to pancreatic dysfunction this procedure would be not illogical However it was proposed as a means of reducing parasympathetic tonus to the colon for the treatment of this disease and such reasoning must be considered totally lacking in logic for the simple reason that the left colon is not innervated by the vagus but rather by the parasympathetic nerves which arise from the sacral division of this system

Diseases of the Autonomic Nerves Themselves

Embryonal tumors of the cells which give rise to the autonomic nervous system are occasionally found usually early in life and are known as *sympathicoblastomas* or *sympathicogoniomas* Tumors of mature autonomic nervous tissue are found in the form of (1) pheochromocytoma a tumor of the adrenal medulla the cells of which originally came from the anlage of the autonomic nervous system and (2) tumors of the argentaffin system known as *argentaffinomas* or *carcinoids* these are not uncommon in the gastrointestinal tract particularly in the ileocecal area and elsewhere where there are accumulations of this type of tissue such as the bifurcations of the carotid artery and of the aorta (organ of Zuckerkandl) The surgical physiology of these interesting tumors is discussed in Chapter 23

SUMMARY OF PRINCIPLES

1 The autonomic nervous system consists of two distinct divisions the sympathetic (thoracolumbar) and the parasympathetic (craniosacral) Each originates from cells in the central nervous system and communicates with viscera by way of two axones a preganglionic and a postganglionic the intervening ganglion in the case of the sympathetic system is usually in a series of ganglia located paravertebrally and the parasympathetic ganglia are in or near the viscera innervated

2 The functions of the autonomic nervous system are adaptive in nature the two divisions are antagonistic and reciprocal to each other Acting together they constitute an important homeostatic mechanism for the maintenance of relative stability in both visceral and somatic functions.

3 The autonomic nervous system is studied extensively by pharmacologic means by the use of mimetic and lytic drugs Other methods of study include surgical extirpation of various portions of this system followed by studies of visceral and somatic function

4 Sympathicotonic diseases are those in which there is a sympathetic hypertonus for reasons sometimes obvious and at other times obscure

5 Raynaud's phenomenon is a symptom complex in which there is marked vasoconstriction of the peripheral arteries most commonly of the

was formerly performed have disappeared from the list of indications for sympathetic denervation as their pathogenesis has become more clearly defined, thus making other types of surgical intervention more logical and beneficial Such has been the case with congenital megacolon (Hirschsprung s disease) which was presumed to be due to an imbalance between the two portions of the autonomic nervous system with dominance of the sympathetic division The results of sympathectomy were not impressive It is now known from the work of Swenson (discussed in detail in Chapter 23) that the pathology of the disease is the absence of intrinsic ganglia of the colon, i e., the plexuses of Auerbach and Meissner Proper surgical therapy now involves resection of the aganglionic segment together with the constriction ring at its distal end

Sudeck s atrophy is the term applied to the changes in bone following trauma particularly with disuse of the part. It has been suggested that the demineralization of the bone is due to a vasoconstriction of the nutrient vessels to the structures involved Results of surgical sympathectomy are equivocal

Parasympathicotonia (Vagotonia)

Parasympathicotonic diseases are sometimes referred to as *vagotonic diseases* simply because the best known parasympathetic nerve is the vagus. The exact cause of peptic disease of the upper gastrointestinal tract is not conclusively established However the acid peptic factor in this disease can be influenced by stimulation of or ablation of the vagus nerve which is responsible at least for transmission of the psychic factors of gastric secretion The constitutional aberration which *is* the *ulcer diathesis* is not completely understood and certainly includes more than excessive vagal stimulation of the stomach Meltzer and Auer first proposed and accomplished vagotomy for this disease in 1904 In recent years this procedure has been repopularized by the work of Dragstedt. Although the procedure of vagus nerve section is not accepted generally as the primary treatment of peptic disease it does have its place in certain complications of the disease and in a particular age group of patients. This is discussed in detail in Chapter 24

Asthma a symptom complex rather than a disease entity is the noisy and difficult breathing brought about either by constriction of the bronchi or by partial occlusion of their lumina The vagus nerve supplies the bronchi with nerves which cause them to be constricted sympathicomimetic drugs have the opposite effect, i.e of bronchial dilatation It would seem therefore that the surgical approach to this condition would logically be a section of parasympathetic constrictor nerves. The operative procedure that has been tried for asthma however is not at all a vagus section but rather a cervicodorsal sympathectomy consisting essentially of removal of the stellate ganglion It would seem anomalous that the section of those nerves (sympathetic) which cause bronchial dilatation are removed for a disease in which there is not enough bronchial dilatation. Yet the fact that a sympathectomy is performed for some cases of asthma demonstrates the empiricism that surrounds much of autonomic nervous system surgery As it does elsewhere cervicodorsal sympathectomy for asthma merely removes the *afferent link* in the reflex chain that may be involved in the production of asthmatic symptoms Stimuli arising in the lung whether allergic infectious, or hemodynamic in nature are carried centrally by way of the sympathetic nerves, synapse somewhere in the brain stem, and then return through the vagus nerves to produce their characteristic bronchial constriction. Sympathectomy therefore interrupts this reflex chain and is less deleterious than a high vagotomy would be It is very interesting that ulcer diathesis and the symptom complex of asthma frequently coincide

Much light has recently been shed upon the pathogenesis of acute and chronic recurrent pancreatitis (*quo vide infra*) In an attempt to ameliorate the pathologic physiology of pancreatitis surgeons have resorted

10

Peripheral Vascular Diseases Arterial

I. Introduction
   A General statement
   B Anatomy and physiology of the vascular tree
II Nature of the arterial occlusive process: types of occlusion and organs involved
III Methods of examination and study
   A History
   B Physical examination
   C Pharmacologic methods and plethysmography
   D Angiography
IV Forms of arterial disease pathology and diagnosis
   A Atherosclerosis (1) nondiabetic (2) diabetic
   B Arteriosclerosis
   C Thromboangiitis obliterans
   D Raynaud's syndrome
V Therapy of peripheral arterial diseases
   A General measures
   B Direct surgical approach
VI Traumatic arterial injuries
VII Arteriovenous fistulae
   A Congenital vascular malformations
   B Acquired arteriovenous fistulae (1) etiology (2) pathologic physiology (*a*) local (*b*) systemic (3) diagnosis (4) treatment (*a*) historical (*b*) current
VIII Arterial aneurysms
   A Etiology true and false aneurysms pathology
   B Diagnosis
   C Treatment
IX. Arterial embolization
   A Etiology
   B Usual sites
   C Differential diagnosis thrombosis versus embolism
   D Treatment

INTRODUCTION

General Statement

Life and function are dependent upon the circulatory system. The two major components of circulation are combined to provide each body cell with life giving blood in sufficient amounts to supply its oxygen and other metabolic requirements and to remove its waste products The heart, great vessels

upper extremities, usually in young females of unstable emotional disposition When this picture is observed in male patients it is usually a precursor of scleroderma

6 Thromboangiitis obliterans discussed in more detail in Chapter 10 is a disease in which there is an inflammatory reaction in the arteriolar wall and a marked spastic component Although not completely understood the cause may be an allergic vasculitis Spasm of arteries following trauma is due to a reflex hypertonus of the sympathetic nervous system and may be severe.

7 Causalgia is the term for a peculiar burning type of pain and frequently follows crushing injury or incomplete section of a peripheral nerve Relief of the sympathetic hypertonus by sympathectomy usually brings relief of the pain and to a lesser extent, of the associated diaphoresis, but frequently the burning sensation remains.

8 The cause of essential hypertension is not known, but it is believed to be a sympathetic hypertonus by some Surgical sympathectomy for this disease is largely being replaced by drugs which produce a pharmacologic sympathectomy

9 Parasympathicotonic diseases are due to intermittent hypertonus of a parasympathetic nerve usually the vagus. The most common parasympathicotonic disease is peptic ulceration of the upper gastrointestinal tract.

10 Asthma a symptom complex associated with constriction of the bronchioles or partial occlusion of their lumina, is another common parasympathicotonic disease and is frequently seen in association with ulcer diathesis

11 The role played by the vagus nerve in pancreatitis is not clearly understood surgical vagotomy is not indicated

12 Tumors of the autonomic nervous system anlage cells sympathicoblastomas are usually malignant Tumors of mature autonomic nervous tissue include pheochromocytoma and carcinoids

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10 Asthma, a symptom complex associated with constriction of the bronchioles or partial occlusion of their lumina is another common parasympathicotonic disease and is frequently seen in association with ulcer diathesis

11 The role played by the vagus nerve in pancreatitis is not clearly understood surgical vagotomy is not indicated.

12 Tumors of the autonomic nervous system anlage cells sympathicoblastomas are usually malignant Tumors of mature autonomic nervous tissue include pheochromocytoma and carcinoids

SUGGESTED READINGS

WHITE, J C., R H. SMITHWICK, AND F SIMEONE *The Autonomic Nervous System* 3d ed. The Macmillan Company New York, 1952.

WHITE, J C., AND W H SWEET *Pain Its Mechanism and Neurosurgical Control* Charles C Thomas Publisher Springfield, Ill., 1955

results clinical manifestations and therapeutic indications The most common occlusive process is *atherosclerosis* which consists essentially in deposition of fat in and upon the intima of the blood vessels It is primarily a disturbance of the metabolism of fat cholesterol and lipoproteins Deposits of these fatty materials in vessels anywhere in the body result not only in reduction of their lumina, but also in a roughening of the surface which is conducive to thrombosis by facilitating the adherence of cellular elements and of fibrin Since atherosclerosis occurs commonly in early and middle life and in some families more than in others and is apparently related to diet and other variables, it must be considered a disease not a degenerative process The extreme importance of atherosclerotic vascular disease is apparent when it is appreciated that this process may occur in every vessel of the body and is probably responsible for one out of every three deaths in the adult American male population as well as for much morbidity and mutilation as a result of peripheral gangrene

Arteriosclerosis in contrast to atherosclerosis, is largely limited to medium-sized vessels and consists of fibrosis loss of elasticity and ultimate formation of calcific plaques in the media. It is not basically an occlusive process Such medial disease usually eventuates in a systolic hypertension with a relatively normal diastolic pressure and is compatible with normal longevity The finding of pipestem or trachealike peripheral vessels in elderly persons of sixty seventy and eighty years of age is commonplace It must be considered strictly a degenerative process

Disease of the adventitial layer of arteries is exemplified in the pathology of syphilis This infection is characterized by perivascular inflammation with ultimate occlusion of the vasa vasorum and subsequent weakening of the vessel wall, resulting in aneurysm or hemorrhage Other diseases with involvement of the vascular adventitia are those of the hyperimmune states, which include polyarteritis nodosa, lupus erythematosus disseminata and scleroderma-dermatomyositis In thromboangiitis obliterans which is described later there is also a cellular infiltration in the adventitia as well as throughout the other coats of the vessel There are some who believe that this disease, too belongs in the hyperimmune group

Trauma to blood vessels is conducive to intravascular clotting because the cells and fibrin in the blood are more readily agglutinated to injured intima Similar occlusions are occasionally seen in blood dyscrasias with accelerated clotting time and/or increased blood viscosity as occurs in polycythemia in which there is a tendency for the slower moving cells to leave the coaxial stream and adhere to the intimal surface thus forming the nidus for a thrombus

Arterial insufficiency however produced results in *ischemia* and death of the tissues supplied In the brain, vascular disease may lead to thrombosis or hemorrhage Coronary artery atherosclerosis leading to thrombosis, is the cause of myocardial infarction this will be discussed in detail in Chapter 20 Ischemia of the kidneys is a cause of renal insufficiency and an important factor in hypertension Occlusive arterial disease of the vessels of the lower extremities is the major source of ischemia of the legs and will be the subject of the greater portion of the discussion in this chapter Peripheral arterial ischemia is seen with growing frequency and is responsible for ever more suffering, mutilation, and economic loss This is so because advances in the therapy of infections have produced an *aging population* the greater longevity permitting the ultimate development of vascular disease

METHODS OF EXAMINATION AND STUDY

History

The clinical evaluation of the peripheral circulation is often considered to be a difficult process, requiring specialized skills and technics, and the employment of precision

and pulmonary circuit constitute the central division all other arteries veins and lymphatics comprise the peripheral units The vascular system is a common integrator of all functions of the body Circulatory insufficiency may be due to disease of any portion of its intimately interrelated parts In this chapter we shall deal with affections of the peripheral vessels

Anatomy and Physiology of the Vascular Tree

The vis a tergo of circulating blood is derived from systole of the left ventricle Blood is pumped into the aorta and distributed through a series of successively smaller branches to all parts of the body The ultimate subdivision, the capillary permits the metabolic interchange between the circulating blood and the tissues On the venous side of the capillary extracellular fluid rejoins the circulating mass Successively larger veins return the blood to the right heart by way of the superior and inferior venae cavae From the right ventricle the blood traverses the lesser circulation, the pulmonary circuit (flowing through the pulmonary artery) and the lungs (where it is aerated) and returns to the left heart via the pulmonary veins Adequacy of blood supply to distant parts depends upon (1) competence of the pumping mechanism (the heart) (2) maintenance of normal pressure relations in this intricate conduit system and (3) organic and functional patency of the venous return

Collateral circulation denotes the existence of alternate routes of blood supply to a part. Such additional pathways may be accessory only in that they are ordinarily not in use but are called into play when the main routes of supply are blocked Collateral channels are occasionally brought into existence where they did not previously exist the stimulus for development is the metabolic requirement of the tissues deprived of their *nutriment by inadequacy of the pre-existing* main channels Such insufficiency may be the result of disease or trauma The development of collaterals (literally alongside of) occurs in both the arterial and venous components of the vascular tree The development of collateral circulation is aided and abetted by action of the autonomic nervous system as well as by chemical factors

The arterial wall consists of three parts, adventitia, media, and intima The adventitia is a relatively loose fibrous layer which has the essential function of maintaining relationship between the vessel itself and the surrounding structures The media is muscular and is under the reciprocal influence of the two branches of the autonomic nervous system These maintain normal arterial tone as well as mediate changes in the size of the arterial lumina by means of their vasoconstrictive and vasodilative action in response to the variation in circulatory need The aorta does not participate in these changes The intima, or endothelial lining of the blood vessel normally provides a smooth surface for the transport of blood with a minimum of friction In disease it may through proliferation occlude the lumen or through roughness, lead to thrombosis

Circulation to a part may be compromised by changes in capillary physiology due to (1) obliteration of the capillary lumina by the sludging of cells as in blood dyscrasias (polycythemia) and in thermal injuries, and (2) specific diseases which affect the endothelial cells of the capillary (vasculitis) such as necrotizing arteriolitis (glomerulonephritis, malignant hypertension and periarteritis nodosa)

The veins are vessels which return blood from the capillary system to the heart They are thinner walled, more variable anatomically and less subject to vasoconstrictive and vasodilative influences than are the arteries The manner in which blood returns from the capillary bed to the heart is discussed later in this chapter

NATURE OF THE ARTERIAL OCCLUSIVE PROCESS: TYPES OF OCCLUSION AND ORGANS INVOLVED

A variety of disease processes affect the arteries with correspondingly different end

results clinical manifestations and therapeutic indications The most common occlusive process is *atherosclerosis* which consists essentially in deposition of fat in and upon the intima of the blood vessels It is primarily a disturbance of the metabolism of fat cholesterol and lipoproteins Deposits of these fatty materials in vessels anywhere in the body result not only in reduction of their lumina, but also in a roughening of the surface which is conducive to thrombosis by facilitating the adherence of cellular elements and of fibrin Since atherosclerosis occurs commonly in early and middle life and in some families more than in others and is apparently related to diet and other variables, it must be considered a disease not a degenerative process The extreme importance of atherosclerotic vascular disease is apparent when it is appreciated that this process may occur in every vessel of the body and is probably responsible for one out of every three deaths in the adult American male population as well as for much morbidity and mutilation as a result of peripheral gangrene

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METHODS OF EXAMINATION AND STUDY

History

The *clinical* evaluation of the peripheral circulation is often considered to be a difficult process requiring specialized skills and technics, and the employment of precision

instruments and complicated laboratory and roentgenographic methods This is not true A careful history and an orderly physical *examination will* permit the determination of ischemia, its severity and its probable cause by the use of inspection and palpation together with a few simple specific tests which are within the scope of any student or physician Such examinations should not only be applied to every patient with symptoms suggestive of vascular insufficiency but should be included *in the* study of *all* persons who have reached the age where vascular impairment may be anticipated. They require very little time and should become a familiar part of every clinical examination

Physical Examination

A history taken where peripheral arterial *disease is suspected must not only* include the general subjective symptoms and manifestations presented by the patient but take cognizance of the age and sex of the patient, and particularly of cardiovascular renal disease and of diabetes The general physical examination will elicit information relative to these conditions and to the patient s gen*eral body economy The examination is now* specifically directed toward vascular examination of the extremities Exposure of both lower extremities must be complete and at room temperature so that visualization may be unimpaired and comparison between the two extremities reliable One observes first of all the *color* of the skin and any deviations from normal whether pale or livid, cyanotic or reddened *Lividity* a purplish reddish hue is commonly first seen at the extremities of the toes Gross inspection may further reveal *trophic* changes of the skin and the cutaneous appendages. Such changes may include the loss of hair the loss of Langer s lines and the thin, wrinkled cigarette-paper like texture of the skin of the leg and toes *Ulcerated and necrotic areas* may also be visualized Color changes on elevation (*pallor*) and on dependency (*rubor*) can regularly be elicited in the presence of arterial insufficiency

Palpation of the skin of the extremity by the dorsum of the examiner s hand may reveal a *temperature gradient* more steep than normal or more so in one leg than in the other the level of *critical temperature change* is not uncommonly very sharp and will help to localize the site of arterial obstruction. The higher this critical level, the more severe is the ischemia The actual site of arterial obstruction is always higher than the critical temperature level and is usually at the next *higher* bifurcation *Palpation* is next carried out for determining the presence absence or adequacy of arterial pulsations of the lower extremities This is begun with the palpation of the femoral artery just below the inguinal ligament, comparison is made on the two sides If the femoral pulsations are good one next palpates for the popliteal pulses *at the superior end of the popliteal* space In the obese patient palpation of the popliteal pulsation is not reliable The dorsal pedal pulse is found on the dorsum of the foot, usually between the first and second or second and third metatarsals. The posterior tibial arterial pulse is palpated about ½ in dorsal and ¼ in proximal to the *medial malleolus The latter* is usually the more reliable of the pedal pulses The presence or absence of the pedal pulses is of great importance and obvious differences between the two feet are significant. This is true because a very large percentage of arterial occlusions occur at the trifurcation of the popliteal artery into the tibial vessels From this site distally collateral circulation is less adequate than in the thigh at the level of the femoral vessels

Pharmacologic Methods and Plethysmography

Further investigation by a few simple procedures may more sharply define the nature, site and degree of the vascular impairment Reference is made to the use of the *oscillometer* an instrument which is really a segmental plethysmograph in that the cuff wrapped snugly around the calf for instance reflects graphically (in arbitrary units) the

changes in the volume due to the systolic distention of the arteries in that segment Although read only in arbitrary units oscillometry is of value because it permits determination of pulsation between the levels of palpable pulses and because it is more quantitative than digital palpation *Plethysmography* a study of volume changes in an organ can yield much information about the blood supply to that organ This technic is particularly applicable to the extremities and most particularly to a digit The ability to produce volume changes in a digit by pharmacologic means reveals the normal adaptive abilities of the vasculature to that digit, in addition to automatically establishing the integrity of the neuromuscular control mechanism

The ability of blood vessels to dilate when the nervous control of their tone is removed or when hyperemia of a part becomes necessary can be measured by the *temperature changes* in the skin of that part. Peripheral nerve blocks and reflex hyperemia are thus standard tests of vascular adequacy The temperature of the skin of an extremity varies directly with the amount of blood flowing through that extremity per unit of time Diminished blood flow may be due to either *spasm* or *occlusion* of the vessels, or both Spasm or residual dilatability can be demonstrated by eliminating vasoconstrictive influences through peripheral nerve block reflex warming of the extremity spinal or general anesthesia, or the ingestion of vasodilators such as alcohol or nitrites or ganglionic blocking agents (sympathicolytic or parasympathicolytic) Blocking of the sympathetic ganglia themselves by Novocain injections is a frequently used means for producing maximal vasodilatation. If the vascular lumen is blocked by occlusive disease obviously no such increased warmth will result. In thromboangiitis obliterans, where both spasm and occlusion are present, the degree of dilatability can be determined by these means Reflex warming the Landis Gibbons test, is due to a *reflex vasodilatation* of the arteries of the lower extremities produced by placing the upper extremities in hot water The *histamine wheal test* the injection of histamine intracutaneously normally leads to a reactive hyperemia due to this chemical and is another method of demonstrating vasodilatability In vascular occlusive insufficiency the response is markedly reduced and delayed The autonomic nervous system is studied by the same drugs that are used in clinical therapeutic testing Thus the reciprocal function of the two portions of the autonomic nervous system is studied by the use of vasoconstrictive drugs (sympathicomimetic or parasympathicolytic) and vasodilator drugs (sympathicolytic and parasympathicomimetic) Some pharmacologic agents are capable of blocking ganglionic synapses without discrimination between the sympathetic and the parasympathetic.

Angiography

Angiography is the roentgenographic visualization of the blood vessels. By the relatively simple technic of placing needles and/or catheters at proper levels in the vascular tree (aorta and femoral and popliteal vessels) the entire circulatory tree of the extremities may be visualized The widespread use of these technics in the past 10 years has aided in permitting a broader attack upon vascular problems of many types The uses of arteriography have been extended to the study and diagnosis of renal disease and cerebral ischemia Recently unilateral renal disease has been cured by the detection of thrombosis of one renal artery which when removed caused the restoration of normal renal function (Fig 12) Similarly cerebral ischemia has occasionally been corrected by detection of thrombosis of a carotid artery and restitution to normal obtained by appropriate surgical therapy

Finally pathologic examination of the vasculature itself by biopsy of peripheral tissues skin and muscle frequently reveals the pathologic entity with which one is confronted. Such examination may be *definitive* in the differential diagnosis of atherosclerosis obliterans versus thromboangiitis obliterans



FIG. 12. Aortogram in a young patient with *hypertension* of unknown cause. Normal left renal artery with enlarged left kidney Markedly narrowed right renal artery with shrunken right kidney At operation the right renal artery could not be replaced nephrectomy was performed with subsequent disappearance of the hypertension.

and may be critical in the detection of systemic disease with diffuse involvement of the vasculature The latter refers to diseases of the hyperimmune syndrome (polyarteritis nodosa, lupus erythematosus disseminata, etc.)

FORMS OF ARTERIAL DISEASE. PATHOLOGY AND DIAGNOSIS

Atherosclerosis

Nondiabetic Atherosclerosis as mentioned above, is the common form of arterial disease which is responsible for such wide *spread mortality and such diversified mani* festations In the lower extremities it is by far the most prevalent basis of ischemia and, ultimately of necrosis and gangrene The pathogenesis of this disease consists in deposition of lipoid material in and upon the intima, with roughening and ulceration of the surface and deposition of calcium with the formation of plaques in the atheromas. Secondary thromboses are extremely common and they determine the severity of the clinical picture Occlusions of the principal arterial trunks by thrombi bring about acute major ischemia and often massive gangrene On the other hand the narrowing of the vessels may proceed gradually and permit the development of collateral vessels. It must be borne in mind that, associated with the metabolic changes of atherosclerosis, there may be coincidental arteriosclerosis of the degenerative variety

Diabetic In patients with diabetes mellitus there is often a characteristic distribution of the most seriously affected vessels In such instances the small arteries of the feet (digital and metatarsal) are often occluded *by thrombi* or *atheromas while the* larger trunks (dorsal pedal posterior tibial and more proximal) are still patent. The necrosis of tissue resulting from such distribution is quite characteristic. Perhaps because of an increased susceptibility to infection in diabetes the necrosis is ushered in by infection or trauma and in many instances, takes the form of intractable burrowing suppuration In others there is progressive cutaneous ulceration which tends to spread in depth and extent until viability of a large part of the foot is lost. It is likely that the initial trauma or infection may increase the oxygen deficit i e., *may render a relative* ischemia absolute An additional factor is probably the progressive thrombosis of digital arteries resulting from contiguous infection

The end result of untreated peripheral ischemia is necrosis of tissue and gangrene. *Dry gangrene* or *mummification* results when the ischemia is absolute and there is no superimposed infection *Wet gangrene* results from the infection of devitalized tissue secondary invasion by saprophytes is common Dry gangrene is no threat to the pa-

tient's life, since nothing is absorbed. In wet gangrene the superimposed infection may constitute a threat to limb and life.

The diagnosis of occlusive atherosclerotic arterial disease is made from relatively characteristic history and physical examination. The subjective sensation of *coldness* of the feet appears early in the typical history. It is experienced particularly on exposure to a cool or cold environment. The patient also observes the skin of his feet to be cold to touch. *Paresthesia*, the sensation of numbness and tingling, accompanies or follows the temperature sensation. Somewhat later the patient will complain of *pain* in the lower extremity, which is usually experienced early only upon muscular exertion. This pain may be in the thigh, in the calves, or in the feet. The reproducible phenomenon of pain on exertion which is relieved by rest is known as *intermittent claudication* and is the characteristic manifestation of ischemia of muscle. The progression from pain upon effort to pain at rest (claudication decubitus, rest pain) bespeaks an advanced stage of ischemia. The occurrence of otherwise relatively minor infections of the feet and toes, which respond poorly to the usual management and refuse to heal because of the ischemia, is frequent.

Physical examination elicits the typical signs and findings. *Pallor* of the skin is usually obvious; it may be increased on elevation. Dependency of the extremity usually elicits *rubor*. *Trophic changes* in the skin of the lower extremity, which include a loss of hair, change of texture of the skin, frequently with loss of Langer's lines particularly of the toes, are observed. *Lividity* of the toes, the dorsum of the foot, and occasionally the heel is observed. *Atrophy* of the muscles of the leg bespeaks chronicity. Diminution or absence of the *pedal pulses* (dorsal pedal and posterior tibial) is characteristic. The popliteal and femoral pulses must also be palpated, since the arterial occlusion may involve those vessels. The finding of reduced *oscillometric readings* in the thigh, calf, or ankle is corroborative evidence. Associated atherosclerotic findings in other organ systems, such as the heart, brain, and kidneys, can usually be elicited. *Roentgenographic evidence* of sclerotic vessels on scout films is not uncommon. Definitive visualization of these arterial occlusions by the various technics of *arteriography*, including aortography and femoral arteriography, pinpoints the site of pathology or establishes its diffuseness (Fig. 13).



FIG. 13. Arteriogram of a femoral artery which is not completely occluded but which is severely diseased. Arrows point to notching by atherosclerotic plaques.

Arteriosclerosis

Arteriosclerosis is a much more benign disease than atherosclerosis. It is a degenerative process in which there is loss of elasticity

of the media of arteries with ultimate calcification of the wall It is not basically an occlusive process and is compatible with long life Clinically it is characterized by the finding of arteries which are palpable after proximal compression and a systolic hypertension The enlarged heart is a dynamic response to the loss of normal elasticity of the peripheral vessels Arterial sclerotic catastrophes are rupture of the hardened vessel with hemorrhage into the involved organ and formation of aneurysm

Thromboangiitis Obliterans

Thromboangiitis obliterans is an inflammatory disease affecting principally young adult males with involvement of both arteries and veins and sometimes of the corresponding peripheral nerves Its pathogenesis is not agreed upon and there are many gaps in our knowledge about this process Some workers believe that it is a specific allergy to tobacco and thus part of *the hyperimmune syndrome* Whether this is the entire cause or not, all are agreed that tobacco plays an important role The disease is characterized by severe pain and ultimate ulceration with progressive necrosis. The ischemia results in part from an obliterative endarteritis, often with superimposed thrombosis and in part from arterial spasm The venous manifestations characteristically a superficial *phlebitis migrans* may precede the arterial symptoms by a number of years Visceral vessels may also be affected and death may result from coronary, cerebral or mesenteric arterial involvement

Pathologically all stages of the disease process can be found in a given extremity If the thromboses and occlusions of the small vessels occur over a relatively long period of time many collaterals are evident. In the vessel itself the perivascular inflammation and adventitial fibroplasia are evident thickening of the internal elastic lamina and polymorphs in the media are seen Atheromas are characteristically absent

The diagnosis of thromboangiitis obliterans is usually made in a male in the third, fourth, or fifth decades of life There is almost always a history of excessive use of cigarettes The symptoms are essentially those described above for atherosclerosis obliterans (intermittent claudication, coldness, paresthesia), with the same general list of findings (pallor on elevation dependent rubor and diminished oscillometric readings) A history of migrating phlebitis in the veins of the lower extremities occasionally precedes the signs of arterial thromboses by months to years Tachycardia and elevated blood pressure are not uncommon

The element of spasm is pronounced in thromboangiitis obliterans and the degree should be determined as a guide to therapy This is accomplished by eliminating the vasoconstrictor influences and measuring the changes in cutaneous temperature that result. It is important to remember that both normal tone and superimposed spastic constriction are simultaneously abolished This may be done by a variety of methods. The test *thermal reflex dilatation (Landis-Gibbons)* is a useful method of study In it the vasodilatation of the lower extremities is produced by putting the upper extremities in hot water *Peripheral nerve block* (which carries the sympathetic nerve fibers) by local injections may suffice to reveal the vasospasm. The release of the spastic component of the disease is productive of a marked vasodilatation and is measured by the increase in skin temperature Paravertebral lumbar sympathetic block also releases the vasoconstrictor influences and normal tonus, and its effect is measured by a rise in skin temperature *Spinal anesthesia* is probably the most efficient method of producing the same result.

Raynaud's Syndrome

Raynaud's syndrome is a disease of unknown cause occurring in females of an emotionally unstable disposition in the second third and fourth decades of life The outstanding manifestation is the severe blanching of the fingers (any or all) upon exposure to a cold environment or following

an emotional experience There is no basic disease of the blood vessels themselves rather the phenomenon seems to be purely vasospastic. Nevertheless, often repeated and prolonged spasm of the digital vessels may result in ischemia which is sufficiently great to produce ulcerations of the finger tips Treatment consists in the use of drugs to combat the profound vasospasm benefit so conferred is usually of short duration Even surgical sympathectomy of the upper extremities does not offer a permanent cure Psychiatric therapy is adjunctive and useful When the symptoms of this obscure syndrome occur in the male they are referred to as *Raynaud's phenomenon* and usually herald the onset of the collagen-derangement disease known as *scleroderma*

THERAPY OF PERIPHERAL ARTERIAL DISEASES

General Measures

It is to be noted that we have elected to discuss the therapy of all forms of peripheral occlusive arterial diseases together This is because the therapeutic measures are largely the same regardless of the disease In addition, however there are specific measures to be employed in each of the main categories

In the regimen for the prevention of the sequelae of arterial disease there are several modalities of therapy which are advisable regardless of the cause of the disease (1) It is desirable that hygiene of the feet be meticulous this includes cleanliness of the skin in order to avoid infections both of the pyogenic and saprophytic types Warmth and dryness are essential features of good hygiene (2) Prevention of vascular spasm of the lower extremities is important exposure to cold and the use of tobacco are the offenders most to be avoided (3) Vasoconstricting agents must be guarded against, including medicines useful in other diseases (4) Buerger's exercises are mildly helpful they consist in the alternate elevation of the extremities above the level of the heart return to the horizontal, then to slight dependency and back to the original position These are repeated for 15 min several times daily A variation of this exercise is passive elevation and dependency by means of the oscillating bed The desired effect of these exercises is the mechanical filling and emptying of the vascular tree (5) Reflex vasodilatation may be promoted by alcohol or drugs, which are ingested or by the application of heat to other parts of the body It is emphasized that heat must never be applied directly to a limb which is already ischemic such an error will result in the increased metabolic demands by the already ischemic tissues for more blood which cannot be supplied. The result will be a hastening of the processes of necrosis and gangrene (6) Intermittent venous hyperemia by mechanical appliances is advocated by some, but it does not produce dramatic results (7) Chemical blocks of the lumbar sympathetic ganglia are advisable particularly if a spastic component can be shown to exist (8) Lumbar sympathectomy is indicated in any case in which a spastic component can be demonstrated. Such is frequently the case in thromboangiitus obliterans. Sympathectomy is of much less use in atherosclerotic occlusive disease specifically because the spastic component of this disease is minimal Occasionally however enough existing collaterals are opened by such a procedure to make it worthwhile It is the consensus that muscle pain alone is not an indication for sympathectomy Skin change including atrophy failure of relatively minor infections to become healed and frank necrosis of skin (gangrene) is the indication par excellence for sympathetic ganglionectomy (9) The therapy of ischemic neuritis is, at best, unsatisfactory A high dosage of the vitamin B complex is useful

Direct Surgical Approach

The most spectacular surgical advancements in the past decade have been in the treatment of peripheral occlusive arterial dis-

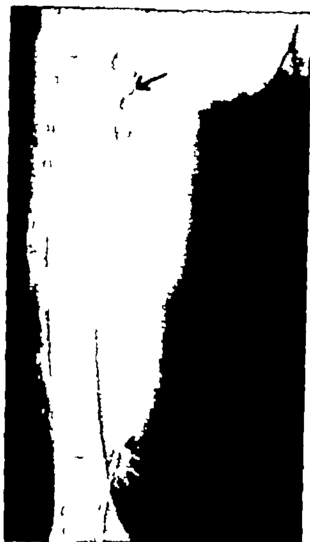


FIG. 14 Arteriogram showing segmental occlusion of the femoral artery Treated with a long plastic bypass.

ease If arteriography of the lower extremities demonstrates the process to be so diffuse that it involves all arteries of the lower extremities, no operative procedure other than possible sympathectomy is indicated If however arteriography indicates that the occlusive process is segmental in nature even though such segments may be multiple several new therapeutic avenues of approach are open *Thromboendarterectomy* the removal of a segmental thrombus, together with the underlying intima in that portion of the vessel is a useful and occasionally limb-saving procedure *Replacement* of the occluded arterial segment has attained even wider application After proper exploration, which determines the nature and extent of the disease as well as the presence or absence of collaterals, the occluded segment may be replaced by any one of a number of materials (Fig. 14) Historically homologous arteries properly removed and stored in artery banks, were the most useful during the early part of this work Except in unusual circumstances their use is now obsolete Autologous vein, usually the greater saphenous or the superficial femoral vein, may be used as a replacement for vessels of the lower extremity They must be reversed in direction so that the venous valves will not obstruct the arterial flow in their new position Their tendency to aneurysmal formation limits their usefulness. Synthetic prostheses of many substances are available for use as replacement vessels The most desirable and successful materials are plastics known as Teflon and Dacron (There are disadvantages and advantages to each of many prosthetic appliances but this discussion does not fall within the scope of this chapter) In order not to disturb the relatively few but highly important collateral vessels in and around an occluded segment, the replacement vessel may be used as a *bypass* rather than as a substitute In such a procedure the prosthesis is anastomosed to the affected vessel proximal and distal to the arterial occlusion, thus bypassing the obliterated area Not only may the vessels of the lower extremity thus be replaced or bypassed, but also those of the upper extremity the carotid vessels, the renal vessels, and the bifurcation of the aorta (Fig 15) The chief requisite in the use of replacement or bypass prostheses so that a high percentage of successes may be anticipated is the adequacy of the outflow vascular tract Diffuse distal occlusions presage failure

Amputation of the extremity is a last resort and is necessitated usually by major tissue necrosis One falls back upon this operation only if none of the preceding methods is effective or if other coexistent diseases make extensive vascular replacement unwise The site of election is dependent upon the level of ischemia the nature of the disease and the usefulness of the re-

maining portion of the limb this is discussed in Chapter 7 When tissue necrosis is gross, and particularly when the gangrenous area is surrounded by an area of cellulitis *refrigeration* of the extremity serves two useful purposes Of primary importance it slows the absorption of toxic products of tissue breakdown and infection which may be a threat to the patient s life Time so gained and used for support of the patient with blood fluids, and antibiotics and in the management of an uncompensated diabetes may weigh in favor of the patient s ultimate recovery Additionally refrigeration may be the only form of anesthesia required in the elderly and debilitated patient

Many patients suffering with diabetes have associated atherosclerotic arterial disease They present themselves with necrotic lesions of the feet in general about a decade earlier than the nondiabetic with the same degree of tissue death Management of diabetes and intercurrent infection demands meticulous simultaneous attention In certain respects the prognosis is better in the diabetic patient than in the pure atherosclerotic. As mentioned earlier in this chapter in the section Forms of Arterial Disease Pathology and Diagnosis, they frequently have occlusions of only the digital and the metatarsal vessels with preservation of patent dorsal pedal and posterior tibial vessels. Why this is true is not known however because of it, relatively minor amputations are possible in the diabetic at a stage where such conservative management is not indicated in the usual form of atherosclerosis obliterans For example, the transmetatarsal amputation (recently advocated by McKittrick) is applicable and useful in young diabetics whereas the use of such an operation in a nondiabetic with diffuse atherosclerosis of the leg will result in failure to heal.

Ultimately in years to come, the treatment of all atherosclerotic disease including that of the extremities probably will be nonoperative The dawn of such a day will be heralded by the disclosure of the metabolic



FIG. 15 Aortogram showing obst (atherosclerosis) of the right commc artery and plaques in the left comm artery Note also the renal arteries and gram as well as the *inferior mesenteric* and all its branches.

At that time the prevention of athe rosis and its sequelae will be a major in the prolongation of life and in the of many limbs.

TRAUMATIC ARTERIAL INJUR

Direct trauma to arteries, either b force (crushing) or by laceration, d the most meticulous care Maximal of *arterial spasm* are produced by tra the vessel with resulting complete ation of the lumen distal to the site of A cold and pulseless extremity first the emergency room, is not to be fors hopeless on casual inspection and su to amputation. Such injuries must

patient seeking medical help is systemic, owing to the pathologic physiology of arteriovenous aneurysm since these lesions produce widespread hemodynamic effects. The systemic findings may include (1) a lowered systemic diastolic blood pressure, (2) tachycardia, (3) the positive auscultatory finding of a bruit over the site of the fistula, a constant accompaniment of such an arteriovenous lesion and characteristically present throughout the cycle with systolic accentuation (4) the signs and symptoms of cardiac decompensation and (5) clinical and laboratory evidence of an increased circulating blood volume (Fig. 17) Digital pressure over the artery proximal to the fistula is rapidly followed by an increased systolic and diastolic blood pressure and a lowered heart rate removal of the digital compression restores the tachycardia and diminished pressure This is known as *Nicoladoni Branham sign*

Treatment Recent advances particularly those made in the Armed Forces during World War II and the Korean conflict, have revolutionized the management of fresh arterial injuries and have been reflected in a profound reduction in the late sequelae In line with these experiences, the prophylactic treatment of arteriovenous fistulae is essentially early and thorough *exploration* and *repair* of arteries in the initial surgical therapy of wounds Such injuries must be recognized and treated per primam by suture or replacement Adequate exposure blood replacement modern armamentarium and refined technic are essential in preserving circulation and preventing loss of life and limb

The definitive treatment of arteriovenous aneurysms has also undergone great change in recent years The most commonly employed operation in the past was quadruple ligation and excision of the fistulous tract, with resultant circulatory insufficiency of the extremity in an appreciable percentage of cases Quadruple ligation was delayed until collateral circulation had been given an opportunity to develop These *collaterals* usually developed after 6 weeks, particularly if

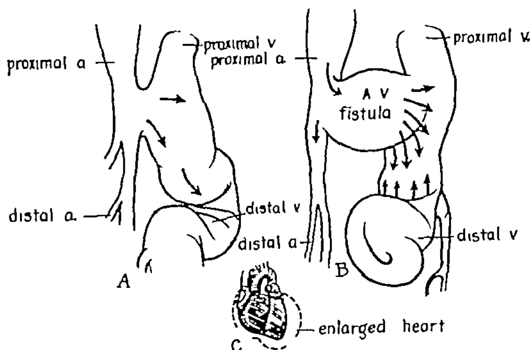


FIG. 17 Arteriovenous fistula *A* The developing pathologic anatomy with the proximal artery larger than the distal and the distal vein becoming larger than its proximal portion *B* A later stage with gross exaggeration of all findings *C* The progressive cardiac enlargement which is a part of the pathologic physiology

intermittent compression of the artery above the fistula was practiced for purposes of stimulation of such collateral growth Lumbar or dorsal sympathectomy was useful in opening the collateral bed as widely as possible before the quadruple ligation was accomplished Matas operation (endoaneurysmorrhaphy) for obliteration of the abnormal communication from within was ingenious in its time but is now of historical interest only In recent years the operative therapy of arteriovenous fistulae has become truly definitive in that they may now be excised and replaced by appropriate grafts. As in the case of *replacement* for occlusive arterial disease such grafts may be of autologous vein homologous artery or most usefully today synthetic materials When the A V communication is obliterated the pathologic physiology and all the clinical findings that it produces are quickly reversed

ARTERIAL ANEURYSMS

Etiology: True and False Aneurysms; Pathology

Arterial aneurysms are dilatations of the vessels resulting from weakening of the walls by disease or trauma. Traumatic vascular aneurysms are true or false lesions A *true aneurysm* is one which has an actual vascular wall even though it be a diseased one A *false aneurysm* is a blood-filled cavity in tissues which is in communication with the vessel lumen through its injured wall and which is surrounded in time by a fibrous sheath The pulsation may be transmitted from the vessel which it overlies The diseases most commonly found as pathogenetic agents of true aneurysms are (1) arteriosclerosis with degeneration of the medial wall, of moderately large-sized vessels and (2) syphilis, in which disease the perivascular inflammation is responsible for obliteration of the vasa vasorum Aneurysms are either saccular or fusiform in character Mycotic aneurysms are less common In arteriosclerosis *dissecting aneurysms* may also occur when there is a tear in the intima of the vessel Dissecting aneurysm is one in which the blood has entered between the intima and the media and separated these two layers the space between these two layers can be enlarged to massive proportions

Diagnosis

The diagnosis of an arteriosclerotic arterial aneurysm is made on the history of a mass appearing along the course of an artery in a patient who is usually elderly and in whom markedly hardened peripheral vessels can be palpated The most common site for peripheral aneurysms is the popliteal artery The aorta and other large peripheral vessels are also not infrequently involved. In addition to producing pain in the area of the aneurysm owing to involvement of adjacent nerves, the arterial supply to the extremity may be further involved by associated intravascular thromboses with ensuing varying degrees of ischemia and gangrene Additional findings are the auscultatory bruit which is present during systole only Compression proximal to the aneurysm does not change the pulse rate or the blood pressure as it does in A V aneurysm.

Treatment

The therapy of arterial aneurysms is again divided into that of the recent past, now of historical interest only and the current therapy Until a few years ago treatment of choice was proximal and distal ligation, with removal of the portion of artery containing the aneurysm. Under such conditions not a few limbs were lost, particularly following interruption of the popliteal artery Other methods used were intraluminal blood coagulation with wires introduced from without and Matas operation of obliterative endoaneurysmorrhaphy or reconstructive aneurysmorrhaphy The current therapy is the same as the treatment of obliterative arterial disease or of arteriovenous aneurysm It consists either in replacing the segment of vessel containing the aneurysm or in bypassing

the area of aneurysmal dilatation with appropriate autologous, homologous, or plastic materials

Aneurysms of the aorta, untreated, may rupture into the pericardium, with death ensuing rapidly from tamponade or into the pleural space with massive hemothorax, or occasionally into the mediastinal viscera esophagus, and/or trachea Aneurysms of the large vessels of the neck produce signs and symptoms referable to (1) central nervous system dysfunction and (2) nerve signs from compression of nerves in the neck. The differential diagnosis of these lesions must include all masses of the neck, and particularly carotid body tumors at the bifurcation of the carotid artery Intracranial aneurysms may simulate brain tumor and must be included in that differential diagnosis.

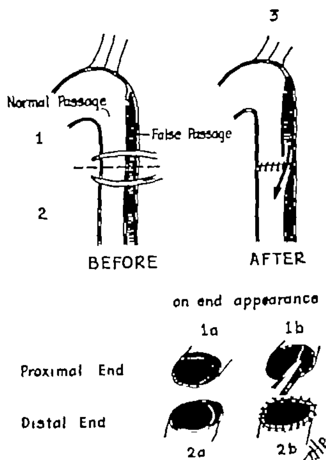


FIG 18 De Bakey re-entry operation for dissecting aneurysm of the aorta Aorta is transected window for re-entry is made in proximal aorta by excising a portion of the inner wall the two walls are sutured together in the distal segment two ends are then reanastomosed.

Arterial aneurysms at the base of the brain frequently rupture and give rise to meningeal signs and to the finding of subarachnoid hemorrhage

A recent chapter has been written in the annals of arterial surgery with the correction of dissecting aneurysm of the aorta. The operation consists in division of the aorta and obliteration of the dissected space between the media and the intima by suturing these two portions of the wall together This, when the aorta is reconstructed constitutes a re-entry portal for blood which has dissected between the walls of the aorta proximally to be directed into its natural lumen distally (Fig 18) Since life expectancy is so short after this lesion is well established this operation is performed as early as possible after the diagnosis is made

ARTERIAL EMBOLIZATION

Etiology

Thus far we have seen that the arterial blood supply to an extremity or to an organ may be obliterated either by occlusive disease of the intima or by aneurysmal dilatation of such a vessel with intravascular thromboses due to roughening of the intima in the presence of eddies and currents Another cause of occlusive arterial disease is that of *arterial embolism* Embolic occlusion occurs when a clot or thrombus migrates from the left side of the heart into the aorta and lodges in any of its branches this may be at any distance from the site of its origin Most clinical cases of arterial embolization have their origin in one of three main disease processes (1) In auricular fibrillation the left auricle is beating so fast that it contributes little if anything toward the filling of the left ventricle Under these conditions, owing to the eddies and currents present in the atrial chamber thrombosis is common in the atrium and its appendage If not firmly adherent to the atrial endocardium, a fraction of such a thrombus may become dislodged and pass through the mitral valve into

ally be preserved by conservative management, the restoration of adequate circulation by operative therapy yields better long-term results

The differential diagnosis of arterial and venous thrombosis and embolism of the intestinal tract is discussed in Chapter 28

SUMMARY OF PRINCIPLES

1 A variety of disease processes affect the arteries with correspondingly different end results clinical manifestations, *and therapeutic indications*

2 The most common occlusive vascular process is that of atherosclerosis, consisting of a deposition of fat upon the intima It is primarily a disturbance of the metabolism of fat and lipoproteins This process may occur in any vessel of the body and produces ischemia to the involved organ

3 Arteriosclerosis is largely limited to medium-sized vessels and consists of fibrosis and loss of elasticity with the formation of calcific plaques in the media It is not basically an occlusive process It may be considered a degenerative process rather than a disease

4 *Involvement of the vascular adventitia occurs in the hyperimmune* states which include the collagen group of diseases and thromboangiitis obliterans

5 Trauma to blood vessels is productive of a maximum degree of spasm distal to the site of injury Vascular occlusion may occur in certain blood dyscrasias with accelerated clotting time and/or increased blood viscosity

6 Because all tissues depend upon adequate oxygenation through competent vascular channels a history taken when arterial disease is suspected must be very extensive

7 *Examination for vascular diseases includes* careful observation of color of skin presence of trophic changes lividity and other signs of adequacy of nutrition

8 Palpation aids in determining the adequacy of arterial blood supply Critical temperature gradients must be localized, and pulses must be palpated Oscillometry is an aid in determining adequacy of arterial pulses. Plethysmography a study of volume changes in an organ can yield additional information about the blood supply to that organ

9 Arterial insufficiency on the basis of vascular spasm is determined by the ability of an extremity to respond to reflex vasodilatation by pharmacologic means and by the removal of sympathetic hypertonus

10 Angiography is the roentgenographic visualization of blood vessels. This technic may be used to study the lumina of the vasculature to the extremities the head and the kidney and of the aorta as well

11 Ischemia of the lower extremities is usually manifested clinically by the progression of the following signs paresthesia coldness pain and intermittent claudication Physical examination reveals pallor of skin trophic changes lividity atrophy of muscles, diminution of pedal pulses and oscillometric readings and arteriographic evidence of diminished blood supply

12 Thromboangiitis obliterans is an inflammatory disease of unknown pathogenesis in which there is a maximal element of vasospasm It is possible that this disease is related to the hyperimmune syndrome.

13 Raynaud s syndrome is a disease of young females of an emotionally unstable disposition it is not primarily an organic vascular disease When this phenomenon occurs in males it may be considered as an early manifestation of scleroderma

14 In the therapy of peripheral arterial diseases general measures include the maintenance of good hygiene the prevention of vascular spasm and other vasoconstricting influences and the removal of sympathetic hypertonus by pharmacologic and surgical means

15 The direct surgical approach to the treatment of peripheral vascular diseases has made great strides during the past decade Added to surgical therapeutic armamentarium are the operations of thromboendarterectomy replacement of occluded segments of the vasculature and bypassing by appropriate prostheses of such occluded segments Amputation is a last resort since every effort is directed toward preservation of tissue

16 Frequently conservative therapy is more easily applied to the diabetic atherosclerotic than the nondiabetic atherosclerotic since the former has a tendency to have occlusive arterial disease of the small (digital) vessels whereas the latter may have a very diffuse distribution of the occlusive process to and including the femoral iliac vasculature

17 Traumatic arterial injuries must be carefully examined with the best of facilities and torn blood vessels must be reconstructed or replaced with synthetic materials

18 Arteriovenous fistulae may be congenital or acquired The congenital vascular malformations include several types of angiomas which may be somatic or visceral

19 Acquired arteriovenous fistulae are abnormal communications between an artery and an adjacent vein usually caused by penetrating or perforating high velocity missiles In the healing process a communication is established between the two channels. Serious local and systemic hemodynamic changes follow The local changes include an edematous extremity dilated tortuous veins and a continuous bruit with a systolic accentuation over the site of the fistula Systemically the early findings include a drop in systemic blood pressure particularly diastolic and a tachycardia. Later there is an increased circulating blood volume tachycardia hypertrophy of the heart, and, ultimately dilatation of the heart with congestive heart failure

20 Compression of the site of an arteriovenous peripheral fistula immediately reverses the findings of the lowered diastolic pressure and the tachycardia. This is known as *Nicolodoni Branham sign*

21 The treatment of arteriovenous fistulae falls into two phases. Prophylactically early and thorough exploration with repair of arteries at the initial surgical therapy of wounds will prevent the development of *many* such

PLATE I

(*A* and *B*) Thermal injury by freezing caused by direct contact with ice and snow for several hours. The hand of a seventy four year-old man who fell down on the ice (*A*) One day after the accident. (*B*) Ten days after admission showing mummification of the fingers.

(*C* and *D*) Thermal injury by burns caused by hot frying oil (*C*) after the fourth group of split thickness grafts. (*D*) After the seventh group of grafts. Early in the postburn period two sets of homologous grafts were used for temporary cover· they lasted long enough (23 and 11 days) to permit healing of the patient's second degree areas, which were then used as donor sites. In addition, the homologous grafts reduced the protein loss from the denuded surfaces.

(*E* and *F*) Gangrene of toes and part of the foot in a young diabetic man who neglected his management, and whose personal hygiene was poor (*F*) The foot after rigid hospital care of the diabetes and conservative surgical therapy (removal of two metatarsals and all toes this was later converted into a transmetatarsal amputation)

(*G*) Replacement of occluded iliac and femoral arteries (atherosclerosis) by a plastic vessel between the aorta and the femoral artery

(*H*) Thromboendarterectomy of a femoral artery

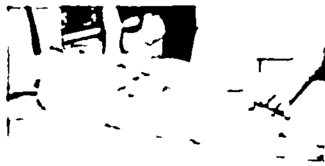
PLATE I





B





D





F





H



I

PLATE II


A


B


C


D


E



F


G


H


I

PLATE II

(*A* and *B*) Coarctation of the aorta in a thirty-six year-old man. (*A*) reconstructed aorta with a 16-mm orifice at the site of reconstruction Note the vagus and recurrent laryngeal nerves being retracted and the left subclavian artery (surrounded by a rubber tape) which is markedly enlarged. (*B*) The 2 mm opening of the coarcted segment from the distal end of the segment of aorta about to be resected.

(*C*) Severly stenotic and calcified aortic valve of rheumatic fever heart disease The orifice seen here from the left ventricular aspect, was not more than 3 mm in diameter the valve cusps were unrecognizable being a bony hard mass varying in thickness from 8 to 18 mm

(*D*) Pulmonary abscess cavity and wall in surgical specimen removed from the right lower lobe of an adult male. Diagnosis could not be made before resection pathologic and microbiologic study proved this to be a case of nocardiosis.

(*E*) Strangulated, gangrenous small intestine caused by adhesive bands traceable to an operation performed 23 years previously

(*F*) Intraductal papillomata of the breast, brought to patient's attention by bloody and grumous discharge from the nipple.

(*G*) Pulmonary abscess cavity and wall resected from the right lower lobe of a seven-month-old girl. This is a specimen from the case represented by the x-ray in Fig 35 in the text.

(*H*) A Meckel's diverticulum in which the lumen was occupied by two faceted fecoliths.

(*I*) Surgical specimen (total colectomy) of polyps throughout the bowel from cecum to rectum.

abnormal communications. The definitive treatment of arteriovenous fistulae has undergone great changes in recent years It no longer consists in quadruple ligation or endoaneurysmorrhaphy The definitive repair is the resection of the area of the A V communication and the restoration of both arterial and venous channels with appropriate materials

22 Arterial aneurysms are dilatations of vessels as a result of weakening of the walls by disease or trauma A true aneurysm is one which has an actual vascular wall even though it be a diseased one A false aneurysm is the extravascular accumulation of blood in a cavity surrounding a vessel, and frequently transmitting the pulsation of that vessel

23 The most common cause of arterial aneurysm due to disease is arteriosclerosis the degeneration of the medial wall There may be a tear between the intima and the media, in which case the blood may dissect between these two layers producing a dissecting aneurysm Therapy is replacement of the segment of involved diseased artery with an appropriate prosthesis Dissecting aneurysms are repaired by closing the abnormal communication between the lumen and the space between the intima and media, thus creating a re-entry channel

24 Occlusive arterial disease is occasionally the result of arterial embolism Such catastrophes occur in the presence of auricular fibrillation and following myocardial infarction in the presence of a left ventricular mural thrombus The sites of arterial embolization are almost always at vascular bifurcations These catastrophes constitute surgical emergencies and embolectomy must be performed soon after the embolization The symptoms produced by arterial embolization depend upon the function of the organ embolized In the extremities the cardinal signs are paresthesia, pain pallor and paralysis.

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Peripheral Vascular Diseases Venous and Lymphatic

I **Physiology of return venous flow**

II **Varicose veins**

A Etiology (1) stock (nature of tissue) (2) increased hydrostatic pressure

B Pathologic physiology

C Diagnosis (1) tests (2) venous pressure (3) venography

D Complications

E Treatment

III **Thromboembolic disease**

A Etiology—predisposing *factors (1) stasis of blood (a) clinical* examples (*b*) pathologic physiology (2) intimal damage (*a*) clinical examples (*b*) pathologic physiology (3) decreased intra vascular clotting time (*a*) hemoconcentration (*b*) polycythemia (*c*) carcinomatosis (*d*) other

B Pathologic physiology (1) *white thrombi (thrombophlebitis)* (2) *red thrombi (phlebothrombosis)*

C Diagnosis (1) thrombophlebitis (2) phlebothrombosis

D Treatment (1) prophylactic (2) direct

E Complications (1) pulmonary embolization (*a*) statistics (*b*) diagnosis (*c*) treatment (*d*) mechanism of death (*e*) fat and air embolism (2) late postphlebitic sequelae the postphlebitic leg

IV **The lymphatic system**

A Anatomy and physiology

B Lymphangitis

C Tumors of lymph vessels

D Injuries and obstruction thoracic duct injuries

E Lymph nodes (1) anatomy and physiology (2) adenitis (3) tumors

PHYSIOLOGY OF RETURN VENOUS FLOW

Veins are thin walled endothelium-lined tubes which return the blood from the peripheral capillaries to the heart Those of the extremities are assisted by valves which project from the walls into the lumina. Veins are distensible and are under vasoconstrictive and vasodilative influences, but *much* less so than arteries

The anatomy and physiology of veins are part and parcel of the mechanism of blood return from capillary beds below the level of the *heart toward* and to the right auricle Since the *arterial head of* pressure from the left ventricle (*vis a tergo*) is dissipated at the capillary level mechanisms of propulsion

of blood from the capillary beds to the right auricle constitute the *venous heart* It consists of three main components (1) the negative pressure in the pleural space which becomes increasingly negative on inspiration and serves to aspirate blood from below the diaphragm toward the heart (2) the squeezing action of the muscles of the lower extremity which aids in propelling blood centripetally (3) the presence of valves in the veins of the lower extremities, which act as rungs on a ladder preventing the column of blood from dropping with gravity Interference with any of these mechanisms will permit the accumulation of fluid in the extracellular space because of the increased hydrostatic pressure at the venous end of the capillary

Venous return from the lower extremities is subject to the added difficulty of propulsion against gravity There are two principal venous systems, the deep and the superficial, which communicate with one another through the perforating veins All these vessels possess valves which permit blood to flow from the superficial to the deep and from the periphery toward the heart. Thus, blood from the foot may enter the deep veins directly or may flow upward in the saphenous system entering the deep circulation at the fossa ovalis, the popliteal vein or any of the communicating veins

VARICOSE VEINS

Varicose veins are dilated and/or tortuous veins. They are most common in the superficial vessels of the lower extremities the greater saphenous system is involved about ten times as commonly as the lesser saphenous system Dilatation of the veins usually results in incompetence of the valves Hemorrhoidal and esophageal veins also frequently become varicose Their special problems are discussed in Chapters 22 and 31

Etiology

The pathogenesis of varicose veins includes essentially two factors (1) *stock* the nature of inherited tissue and (2) chronically increased hydrostatic pressures There are some persons who are predisposed by hereditary stock to varicose veins. In general this is the body habitus which predisposes to varicose veins, hemorrhoids, pendulous breasts visceroptosis, and diastasis recti abdominis Factors which result in an increase in the normal hydrostatic pressure to which the vein walls are subjected include (1) *posture* e g standing for long periods of time while at work, without using the muscles of the lower extremities (2) *compression* from above which hinders the venous pump in its fight against gravity the most common causes are pregnancy ascites, obstruction of the iliac veins and the vena cava by metastatic malignancy or primary tumors within the abdominal cavity or the retroperitoneal space and constrictive clothing worn either on the extremities or as girdles around the abdomen (3) defects of the valves of the veins of the lower extremity whether congenital, mechanical or inflammatory In patients who have had deep vein phlebitis (iliac, femoral popliteal) two factors are produced which cause dilatation of the superficial veins. The permanent damage to the valves of the veins leads to venous insufficiency in the deep system when the patient is standing (Fig. 19) The increased

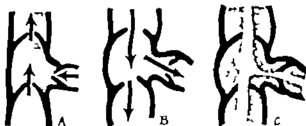


FIG. 19 The venous valves. *A* Normally competent valves, permitting unidirectional flow toward the heart. *B* Dilated veins with incompetent valves (varicose veins) *C* Inclusion of valves in the organization of a prior thrombosis rendering the valves functionless (postphlebitic syndrome)

hydrostatic pressure is transmitted to the superficial veins and they may become dilated Simple *compensatory dilatation* of the

uperficial veins does not constitute varicose eins If however the valves become in ompetent because of the dilatation of the uperficial trunks or if they are damaged by he inflammatory process true varicosities levelop These are known as *secondary vari ose veins*

Pathologic physiology

The pathology of varicose veins includes evidence of intimal damage by mechanical or bacterial trauma and a thickening of the venous wall (*phlebosclerosis*) Evidence of old perivenous inflammation is common The deep veins are protected by muscles the superficial veins lie in yielding fat The essential pathologic physiology in varicose veins is incompetence of the valves in the superficial and communicating systems. If those in the saphenous veins alone are nonfunctioning there is relative stasis in these vessels when the patient is standing If in addition, the valvular incompetence includes the communicating system (which usually results from phlebitis) blood from the higher-pressured deep veins is squeezed back into the superficial system Under these circumstances venous insufficiency and edema supervene

Diagnosis

The diagnosis of varicose veins is usually made on visualization of large and tortuous superficial veins of the lower extremities The patient usually complains of tiredness and heaviness in the legs with or without accompanying edema Not infrequently the varices are totally asymptomatic relief is sought for cosmetic reasons Skin changes, including atrophy of skin, pigmentation and induration of the skin and subcutaneous tissues, and even ulcers are occasionally seen as sequelae to simple varicose veins. However it must be emphasized that such changes are usually part of the associated postphlebitic syndrome a sequel to deep occlusion, which will be discussed later in this chapter The common area of skin involvement when the greater saphenous system is primarily involved is just proximal to the internal malleolus whereas with the involvement of the lesser saphenous system it is just proximal to the lateral malleolus The diagnosis is further made and refined by (1) palpation of the saphenofemoral junction for appreciation of its incompetence (2) the Trendelenburg test and (3) the Perthes test. In the Trendelenburg test the incompetence of the valves of the saphenous system is established. The leg is elevated above the level of the heart and the tourniquet is applied on the thigh When the patient stands up if the saphenous system valves are competent, that system will fill slowly from below usually in 30 sec or more If the saphenous system valves are incompetent, the saphenous system will fill almost immediately and from above downward as soon as the tourniquet is removed If the valves in the communicating (perforating) veins are incompetent, blood will immediately flow from the deep veins into the superficial distending them while the proximal tourniquet is still in place The Perthes test attempts to establish the patency of the deep venous system A tourniquet is placed around the thigh in the standing position causing the superficial system to be distended with blood. If the patient now walks, the superficial veins will empty if the deep system is patent if the deep system is occluded the superficial system remains distended.

The critical pathologic physiology of varicose veins is a matter of the pressure within the veins and this can be demonstrated clinically In an individual of an average height the distance from the foot to the right auricle is about 120 cm Therefore the pressure within the veins of the foot is equal to the column of blood between the foot and the right auricle (120 cm H_2O or about 10 mm Hg) A needle can be put into one of the superficial saphenous branches and connected to a manometer which can be rolled along on the floor as the patient walks After determining normal venous pressure in the recumbent position pressure in the standing position is then determined. If the deep

venous system is patent, when the patient walks the blood will drain from the superficial system into the deep system and the standing venous pressure will be reduced If however there is deep venous occlusion, the pressure in the superficial system when the patient walks will remain high or even become elevated. This is the concept of *ambulatory venous hypertension* which is the critical pathologic physiology of varicose veins.

The incompetence of the valves of both the superficial and deep systems, and the consequent pooling of blood, can be demonstrated by the technic of *venography (phlebography)* A radiopaque material is injected into a vein of the foot with the patient recumbent on the x ray table with the head of the table elevated about 25° Exposure of films underneath the entire extremity from the toes to the groin will reveal either the normal pattern of venous drainage with competent valves or a marked pooling of blood in either the deep or the superficial systems or both similarly deep venous occlusions before the process of recanalization may likewise be demonstrated This technic is only occasionally resorted to in clinical practice

Complications

The *complications of varicose veins* of the lower extremities are (1) an occasional rupture of a varix with hemorrhage and (2) skin changes including ulceration at the characteristic sites mentioned. Since these skin changes are most characteristic in association with deep venous occlusion they will be discussed later in this chapter under the heading of Complications in the section on Thromboembolic Disease

Treatment

In the therapy of varicose veins of the lower extremities one directs attention first against the underlying disease if such exists. This refers particularly to those which produce compression of the veins in the pelvis and in the abdomen Many patients with varices of the lower extremities are relieved with simple compression bandages applied from the feet to the lower thighs Such compression bandages give support to the venous return and produce relief of symptoms Best permanent results are obtained by the stripping operation, wherein the entire length of the saphenous veins, greater and lesser are stripped from their most accessible distal branch to their entry into the deep system The stripping operation is most easily accomplished in the patient whose leg is not obese in whose leg the vein is relatively straight, and in whom little phlebitis has existed prior to operation Lesser procedures for the surgical treatment of varicose veins are usually followed ultimately by the stripping operation. Where much superficial phlebitis has existed a good stripping operation is not easily accomplished. Where there are collections of tortuous veins these must be excised en masse and the stripping pursued above and below the site of these excisions It is worthy of emphasis that it is necessary to seek out and ligate incompetent perforating veins if they exist. Obliteration of varices by the production of a *chemical endophlebitis* using sclerosing agents such as the soaps of fatty acids, is rarely used as a method of choice today Rather this technic is only occasionally used as an office procedure for minor varices For many years the surgical therapy of varicose veins included the ligation of the greater saphenous vein at its entry into the fossa ovalis and the ligation of all of its tributaries This was accompanied by multiple incisions on the leg and ligation of the saphenous vein above and below its perforating branches with ligation of the communicating branches between the superficial and deep systems. In general the stripping operation is most successful in the therapy of varicose veins and should be used as the method of choice all other technics are expedients

THROMBOEMBOLIC DISEASE

Thromboembolic disease refers to phenomena associated with abnormal intra

vascular coagulation of blood and its pathophysiologic sequelae A *thrombus* is a clot intravascular *thrombosis* is the occurrence of a clot within a blood vessel an *embolus* is a clot (or other material) which has migrated within the vascular tree

Etiology—Predisposing Factors

About 90 per cent of all cases of venous thromboembolic disease begin in the deep veins of the lower extremities the remaining 10 per cent arise in the veins of the pelvis and intestinal tract. The largest part of the discussion in this section pertains to the deep veins of the lower extremities The pathogenesis of thromboembolic disease includes the predisposing factors of (1) *stasis of blood* (2) *intimal damage* (3) *decreased intravascular clotting time*

Stasis of blood means that the return venous flow is slower than normal and that the time that elapses between the passage of blood from the capillary circulation to the right auricle is prolonged The clinical diseases in which this situation obtains are (1) congestive heart failure which constitutes the largest single group (2) any obstruction to the vena cava or the iliac veins as in intestinal obstruction in ascites and where there is compression of these veins from without by primary or metastatic tumors (3) periphlebitis, as occurs in ascending infections and in carcinoma metastases, (4) the presence of varicose veins and (5) clothing constrictions, worn either on the legs or as a girdle around the abdomen

Intimal damage may be of several types infections (bacterial trauma) chronic inflammatory disease which is transmural (thromboangiitis obliterans) or mechanical trauma which produces endothelial tears. The endophlebitis which is produced causes the adherence of platelets to the damaged intima probably because of the release of certain enzymes and results in further precipitation of other blood elements until a clot is formed One is reminded of the student experiment in physiology in which a section of vein is tied off and removed from the circulation and placed on the table Similarly another section of vein is tied off and removed but between the ties it is tapped with a hard instrument and laid alongside the other section of vein Examination of the two sections a few minutes later reveals that the one traumatized with blunt force contains clotted blood whereas the other which had not been traumatized contains liquid blood

Decreased intravascular clotting time occurs clinically in the following diseases (1) wherever *hemoconcentration* is prominent as in dehydration burns and intestinal obstruction (2) polycythemia vera or secondary, (3) carcinomatosis, which produces some changes in blood conducive to rapid clotting as yet poorly understood

Pathologic Physiology

The pathologic physiology of intravascular clots varies with the nature of the predominant cause Thus there are really two main forms phlebothrombosis and thrombophlebitis. *Phlebothrombosis* is characterized by red thrombi which resemble postmortem clots in that they are smooth and contain all blood elements They are characteristic where the predominating cause is stasis usually without initial infection *Thrombophlebitis* is characterized by the formation of white thrombi, in which there is a predominance of platelets and white cells which have been precipitated out and become adherent to the damaged venous intima. They are characteristically found when the predominating cause is intimal damage infectious or traumatic. A *propagating thrombus* is one that continues to grow at its tail end i.e., as it waxes toward the heart in the direction of blood flow Thus it may be seen that, although a process may have started basically as a thrombophlebitis its tail end or central end may more closely resemble the red thrombus and not be adherent to the wall Clinically thrombi rarely occur in these pure forms by the time they are examined surgically mixed thrombi are the rule.

Superficial phlebitis of the saphenous system, is frequently seen with cutaneous in

fections pyogenic or fungous. Superficial phlebitis of nonvaricose veins is rare and usually is indicative of systemic disease such as the *phlebitis migrans* seen in Buerger's disease and in association with carcinoma, particularly of the pancreas *Deep vein phlebitis* may be the result of phlebothrombosis, with its potential serious sequelae It may also be the result of a thrombophlebitis, beginning with an infection, in the post operative or the puerperal period These states are associated with a leg which is markedly swollen and also white owing to the reflex arteriolar spasm resulting in the typical picture of the (*white*) milk leg (*phlegmasia alba dolens*)

With all the awareness of thromboembolic disease and the variety of methods employed in an attempt to avoid it, it cannot be said that the incidence of thromboembolic disease has diminished in recent years This is probably due to the fact that more extensive surgical attacks are being made for diseases only recently considered unapproachable Additionally the average age of patients treated is ever increasing 11 per cent of the entire population is over the age of sixty five Another important factor is that hospitalization for many diseases which were previously treated at home is increasingly common and reflects wide insurance coverage of the population at large Sixty per cent of all thromboembolic disease occurs in medically treated patients 40 per cent in surgical patients this is easily understood when it is appreciated that the outstanding single causative factor in the occurrence of stasis in the veins of the lower extremities is confinement to bed because of congestive heart failure Other factors which predispose to the development of thromboembolic disease are marked general debility carcinomatosis, obesity old age and prolonged immobility as with multiple fractures.

Diagnosis

The diagnosis of *thrombophlebitis* is made when a patient who has recently had an infection in the lower extremities an injury an operation or a delivery complains of soreness and pain in the lower extremity The findings are those of an infection with local and systemic signs. The attack may be ushered in with a chill and there is a rapid rise in temperature. The leg becomes very painful swollen and pale (phlegmasia alba dolens) Redness, induration, and tenderness may be detected along the course of the major vessels The diagnosis of *phlebothrombosis* unfortunately may be extremely difficult to make before a catastrophe has occurred. There may be no local signs whatever or there may be just one an asymptomatic increase in size of the leg, particularly at the calf *Homans sign* pain in the calf on dorsiflexion of the foot, may be an isolated finding There may be no systemic signs whatever or there may be just one a tachycardia with no associated signs or symptoms. An isolated tachycardia is not infrequently an isolated finding when there is some intravascular disease Recurrent episodes of pain in the chest are sometimes understood in retrospect as a shower of emboli to the lung premonitory to a major pulmonary embolism

Treatment

The treatment of thromboembolic disease falls clearly into the subdivisions of *prophylactic* and *direct* In order to avoid the dangers and disabilities of this disease attention must be directed to its prevention This is best done by meticulous preoperative and postoperative care Such care begins with a very careful personal and family history If there have been previous episodes of thromboembolic disease in the patient's past history every precaution must be used to prevent the occurrence of predisposing factors This may include the prophylactic use of anticoagulants. Infections anywhere and particularly in the lower extremities must be cared for before the patient is subjected to operative intervention Obstructive binders either on the extremities or around the abdomen must be interdicted Existing superficial varices must be treated or smoothly

wrapped preoperatively The patient must be instructed in advance that immediately post operatively he is to perform deep breathing exercises and leg exercises at regular intervals this aids in the function of the venous heart and thus prevents stasis Early ambulation is practiced to accomplish the same purpose provided no specific contraindication exists If in the early post operative period especially following thoracic or upper abdominal surgery the patient breathes in a shallow panting manner anesthetization of the intercostal nerves by local injection will relieve him of pain permit deep breathing and aid in venous return from the lower extremities

The direct treatment of thromboembolic disease once it has occurred in the lower extremities depends on the nature of the local disease If the diagnosis is clearly that of thrombophlebitis with its clot adherent to the venous wall there is much less likelihood of embolization Under such conditions the treatment falls into three parts (1) appropriate biotherapeutic agents as for any infection (2) anticoagulants to avoid the formation of a propagating thrombus centrally (anticoagulants do not dissolve an existing clot they only prevent propagation of further clots) (3) relief of the pain which is due at least partly to reflex vasospasm. This can be accomplished by lumbar sympathetic blocks repeated until the vasospasm is abolished

If the diagnosis is phlebothrombosis with its attendant bland thrombus which is not attached to the venous wall the problem is less easily solved Theoretically when the diagnosis has been made of a clot which is not adherent to the venous wall with its attendant danger of becoming an embolus to the lungs the treatment should be *ligation* of that vein somewhere between the clot and the lungs There is no consensus among surgeons about this point There seems to be about an equal division between those who would ligate the superficial femoral vein when the diagnosis of phlebothrombosis in the calf is made and those who would use anticoagulants exclusively In reported statistics there is little to choose in the results between these two alternative methods Most surgeons agree that when the superficial femoral vein is ligated it should be done on both sides because frequently a subsequent embolus has occurred from the side opposite to the one in which the diagnosis has been made Clearly the pre-existing predisposing factors may exist in both legs Occasionally it may be necessary to ligate an iliac vein or upon occasion when the clot has extended into the femoroiliac veins it may even be necessary to ligate the vena cava It is desirable at the time of ligation to open the vein and remove the clot if at all possible If the diagnosis of phlebothrombosis of the lower extremities is made by the catastrophic occurrence of a pulmonary embolization there is little argument against the choice of therapy being ligation of veins bilaterally Suffice it to say that, in addition to ligation, anticoagulants are used

Complications

There are two main groups of sequelae to thromboembolic disease (1) early sequel pulmonary embolization (2) late sequel, the postphlebitic leg

Pulmonary embolization occurs when a fragment or all of a clot in a peripheral vein becomes detached and passes centrally up the vena cava into the right side of the heart, is ejected from the right ventricle and lodges in the pulmonary artery at its bifurcation or in one of its branches within the lung Such an embolus may originate in the right side of the heart, consequent upon the mural thrombosis of myocardial infarction. The transport of many small clots and their lodgment in the pulmonary tissue may occur with only minimal or negligible symptoms and signs Commonly they may be overlooked or misinterpreted with no serious outcome However the transport of a reasonably large fragment of thrombus may occlude the entire blood supply to either a lobe or a lung and cause death or serious illness If one segmental pulmonary artery or lobe

artery is occluded by such an embolus, a segment of lung may or may not become infarcted depending upon the local conditions of that lung If infarction occurs the symptoms and signs will be those of atelectasis and pleuritis with pain and friction rub and will be followed by those of pneumonitis In farction of such a lung, after about 48 hr will show roentgenographic evidence of atelectasis of that segment or lobe In the event that a massive embolus either lodges at the bifurcation of the pulmonary artery or blocks one major pulmonary artery death may ensue instantaneously or rapidly The symptoms and signs are cerebral and cardiac The cerebral signs are those attributable to hypoxia. The cardiac signs, unfortunately are usually fatal and consist of cardiac arrest mediated through the vagus nerves Death from pulmonary embolus cannot be explained by occlusion of the pulmonary artery alone since clamping and ligation of such vessels are routinely done during lung surgery without serious effect Two additional factors (1) vasoconstriction of the remainder of the bronchial arterial tree and (2) vagal influences causing deleterious cardiac action, are invoked in order to explain this catastrophe

When the diagnosis of pulmonary embolization is made therapy must be logical and rapidly applied Atropine preferably intravenously should be administered to avoid vagal reflexes with the all too common cardiac arrest. Oxygen should be administered to avoid hypoxia. Papaverine preferably intravenously is probably the best vasodilator it may permit the passage of an embolus to the smallest possible pulmonary artery branch Anticoagulants should be started soon Appropriate venous ligation must be considered and much will depend upon the conditions of the patient at this time Antibiotics to avoid pneumonitis in an infarcted area are appropriately used Ideally as for embolization elsewhere embolectomy from the pulmonary artery is logical This the Trendelenburg operation, is rarely accomplished, for in the cases where it is most necessary cardiac arrest and death occur most frequently

The *late sequelae* of thromboembolic disease of the lower extremities are embodied in the clinical picture known as the *post phlebitic syndrome* The complaints are (1) *swelling* (2) *bursting pain* (3) disabling *skin lesions* usually of the lower one third of the leg (Fig 20) The swelling of the leg is soft and pitting at first and subsides on recumbency and elevation of the extremity Later it becomes brawny and the skin may be markedly thickened in some areas while thin and atrophic in others. The site most frequently involved is just proximal to either malleolus and extending for a variable distance Within this expanse of abnormal skin (induration inflammation pigmentation) there may be one or more ulcerated areas, which are characteristically painful and indolent. All symptoms are aggravated when the patient is in the upright position and are relieved when several days of bed rest are enforced The chronicity of the infection of this abnormal skin and the tissues surrounding it is striking The edema of such a leg is due primarily to the occlusion of the deep vein in that extremity When recanalization of the deep vein occurs with destruction of its valves by fibrosis an increase of the hydrostatic pressure in the leg results because the vein no longer is an effective part of the venous heart. A second factor may be the protein content of the edema fluid which holds back some fluid in the interstitial space The term *lymph edema* is frequently used implying that the edema is due to obstruction of lymph channels with an outpouring of lymph into the tissues. The role of the lymphatics is generally overemphasized While it may be true that complete occlusion of the lymphatics draining a part may over a long period of time cause edema these conditions do not prevail in the usual case Suffice it to say that venous insufficiency (stasis) is the principal factor and all the manifestations can be explained on this basis

Whereas the pain early in phlebitis is

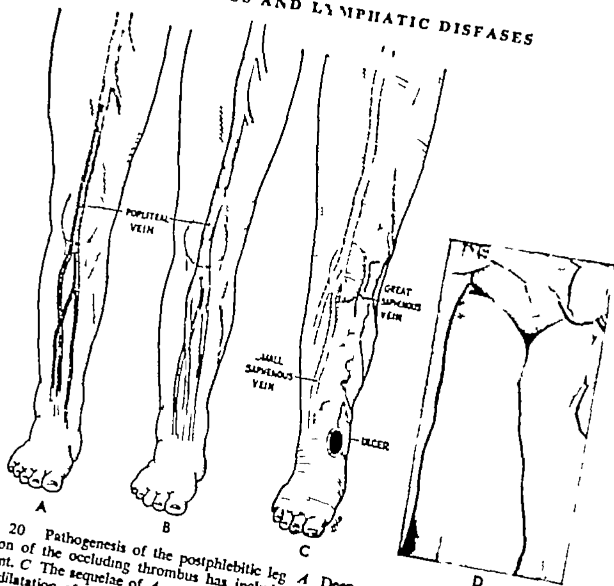


FIG 20 Pathogenesis of the postphlebitic leg *A* Deep venous occlusion. *B* Organization of the occluding thrombus has included the valves and rendered them incompetent. *C* The sequelae of *A* and *B* The leg is edematous and painful, there is secondary dilatation of the saphenous system and skin changes have progressed to the stage where there is an ulcer in the typical supramalleolar area. *D* Occlusion of one common iliac vein, showing the massive edema of the involved extremity extending above the groin.

partially due to reflex vasospasm, this probably plays little role in the chronic state It is more likely due to the pathology of the atrophic, pigmented, ulcerated skin with its chronic, smoldering infection These cutaneous complications of deep vein phlebitis are distressing and disabling and usually not amenable to surgical correction. These changes consist of pigmentation, woody induration, inflammation, and eventual ulceration. The genesis of these changes is still in dispute Stasis alone does not explain them In fact, ulceration frequently occurs in the absence of edema, which is the characteristic manifestation of venous insufficiency It is far more probable that these changes represent a sequence of events beginning with superficial vein phlebitis, followed by perivenous cellulitis and subsequent fibrosis In addition to the chronicity of the infection, the lack of opportunity for collateral venous drainage may be a factor Whereas collateral veins may grow readily in the vicinity of the calf and thigh muscles, there are only skin, fascia, tendons, and bone proximal to the malleoli When such an area is traumatized to a degree that will not damage normal skin a breakdown occurs. Superficial infection supervenes and a localized area of thrombophlebitis occurs

at this point Ultimately with conservative measures such ulcerations usually heal early This process of repeated ulceration and heal ing continues, with repeated infections and scar formation. Finally these patients present themselves with ulcers of the lower leg which bear a marked resemblance to peptic disease of the duodenum Such lesions are surrounded by an area of chronic inflammation and cellulitis, referred to as *lym phangitis streptogenes* As a result of the repeated insults of infection with attempts to heal the skin is more scar than skin. The scarring actually becomes a detriment to further healing, in that there is a constriction of the small arterioles in the area. This pic ture of the late sequelae of the postphlebitic syndrome is a common one in all types of practice is disabling, and constitutes a great economic loss

The treatment of the postphlebitic leg is unsatisfactory at best. Conservative measures are always tried first. These include rest in bed with elevation of the leg slightly above the level of the heart, antibiotics to control the cellulitis and the local infection in the ulcerated area, and compression bandages to help in venous return Caution must be exercised in the use of compression bandages when there is an associated obliterative endarteritis not uncommon in persons of the older age groups Such measures usually yield temporary good results only Excision of the ulcerated area and grafting with autologous skin without attention to the deep veins of the leg will yield most disappointing results These patients should be studied by venography to demonstrate the nature and extent of occlusions of the deep veins In recent years it has become com mon practice to ligate the superficial femoral vein strip out associated varicose saphenous vessels excise the ulcerated and pigmented area of skin to include the large vein with destroyed valves that lies underneath it and *then replace the excised area with split thickness grafted skin.* This extensive procedure yields good results in a high per centage of cases It is doubtful whether ligation of the deep vein adds anything to the efficacy of this treatment. It may be noted that the saphenous system and the deep vein may be removed at one time, this completely disregards the findings of the Perthes test. This procedure has been done in enough places on a great enough number of cases now to warrant disregard of the Perthes test. Indeed, it is the network of collaterals from the muscular branches (*profunda*) which carries the greatest amount of blood toward the heart.

Another late sequel to deep vein phlebitis that has been more commonly recognized in recent years is multiple pulmonary emboliza tions. The throwing off of many showers of small emboli may produce multiple small pulmonary infarctions with the development of chronic cor pulmonale

Phlegmasia cerulea dolens is the spectacular picture seen with the sudden occurrence of a massive thrombosis of the femoral or iliac vein of an extremity The characteristic purplish-blue color of the extremity is due to its suffusion with venous blood which cannot escape from the extremity Under such conditions tissue tension may be so high as to exceed arteriolar pressure resulting in tissue ischemia, necrosis, and wet gangrene The pressure within the venules is raised to such an extent that there is marked di apedesis, a leaking of fluid through the capil laries and smaller vessels into the interstitial space This type of vascular catastrophe is an extreme emergency requiring the com batting of shock and the performance of im mediate thrombectomy

THE LYMPHATIC SYSTEM

Anatomy and Physiology

The lymphatic system begins as a mesh work or reticulum mosaic, of capillary-sized vessels in tissues throughout the body Only the central nervous system the substance of the spleen and muscle bundles do not contain lymph vessels Lymph is collected by this network of lymphatic capillaries from

tissue spaces From the lymph capillaries the lymph is conducted into larger tubular type lymphatics which conduct the tissue juice to the nearest lymph node The lymph is filtered through the lymph node and then proceeds on in efferent lymph vessels to progressively thicker walled vessels until it is ultimately returned to the venous circulation largely through the thoracic duct in the left subclavian vein Other less important points of entry into the venous circuit exist. The tubular lymphatics have valves on their endothelial surfaces which prevent the back flow of lymph Lymph differs very little from blood plasma Ordinarily it contains less protein than blood plasma However in the presence of infection it contains more protein including fibrinogen than the blood under these conditions it clots rapidly The liquid portion of the inflammatory exudate consists largely of lymph. Lymph vessels from the lower portion of the small intestine are adapted for the specific function of transmitting absorbed fats into the lymphatic system and through the thoracic duct into the venous circulation These channels, lacteals are clearly visualized as they carry the chyle in the postprandial state All lymph channels are lined by endothelium

Numerous functions of the lymphatic system are known others are not. Clearly the lymphatic system is a first line of defense against infection from without, since invading material is rapidly carried to adjacent lymph nodes for processes of filtration. Transportation of the largest portion of absorbed fat from the intestinal tract is another major function of the lymphatic system. Additionally the lymphatic system acts as a buffer mechanism in rapid adjustments of fluid and electrolytes, and thus in the maintenance of normal tissue turgor The contribution of the thoracic duct to the venous flow has been variously estimated at 50 to 120 ml per hour

Lymphangitis

Lymphangitis the presence of infection in the lymphatic stream is of two main types. Reticular or capillary lymphangitis is the presence of infection in the fine meshwork of lymph vessels common in the skin This type is seen under the following two main conditions (1) Erysipelas is an infection with *Streptococcus erysipelatis* The lymphatic reticular vessels in the skin become swollen and the infection appears to travel in the skin This is seen clinically as a raised and advancing border of elevation in the skin most commonly seen on the face and forehead however it may occur in the skin of any part of the body The edema of the skin is visualized clearly in the patient as the pores appear like pinholes in a cushion. Antibiotic therapy and no surgical intervention is indicated in this disease (2) Tubular lymphangitis is the presence of an infection in the lymphatic chain of vessels from a peripheral tissue to one or more groups of lymph nodes It is usually consequent upon a tissue injury such as the stick of a pin or any other penetrating injury The infection is usually with *Streptococcus* Clinically red streaks are seen along the course of the lymph channels between the site of injury and one or more groups of lymph nodes draining the area. Surgical intervention is not indicated appropriate antibiotic therapy should be employed

Tumors of Lymph Vessels

Varicosities of the lymphatic channels, *lymphangiectases* are dilatations of lymphatic vessels, probably the result of stasis in the lymph system Treatment is directed at the cause of the stasis. True tumors of lymph vessels are called *lymphangiomas* These tumors may be lymphangioendotheliomas or they may be cavernous in nature The cavernous type of lymphangioma is not uncommon in the neck (cystic hygroma) and is seen as a congenital finding. Excision is the treatment of choice

Injuries and Obstruction Thoracic Duct Injuries

Injuries to the thoracic duct in the neck or in the mediastinum are more common

today because of the more extensive surgical procedures carried out in these areas The thoracic duct courses in the posterior left mediastinum usually from the sixth dorsal vertebra upward It may be injured at this point during dissections of the mediastinum for cardiovascular anomalies or for malignancies within the hemithorax. Likewise in radical neck surgery the entrance into the venous system may be injured. If the injury is appreciated at the time of operation the duct may be ligated with no further untoward sequelae More commonly the injury is not appreciated until the early postoperative period at which time there is a rapid accumulation of fluid in the neck or in the pleural space Aspiration of this fluid will prove it to be chyle Several aspirations of extravasated chyle should be attempted before surgical intervention is resorted to Occasionally such measures will terminate the spillage More often surgical intervention will become necessary for two reasons (1) to avoid disturbance of function, such as compression of the lung or of vital mediastinal or cervical structures (2) to prevent the marked debility that can occur from the loss of fats fluid, and electrolytes in the extravasated chyle If resuture of an injured duct can be accomplished it should be done however simple ligature usually suffices. Collateral channels develop to take over the function of the ligated duct Injury of the *cysterna chyli* a collecting station in the abdomen for all the lacteals, may occur during abdominal surgery Therapy is the same as for injury of the thoracic duct

Obstructions of the lymphatic system may occur anywhere in the body but are most common under two conditions (1) parasitic infestation, most common in tropical areas, usually affecting the lower extremities (2) following radical surgery for cancer or as a result of blockage of lymph nodes by malignancy without surgical intervention A classic example of the former is elephantiasis due to filariae A common example of the latter is the swelling of the upper arm in cancer of the breast with or without dissection of the axillary lymph nodes. (Venous thrombosis may further aggravate the swelling) A chronic form of lymph stasis is seen as a part of the postphlebitic syndrome in the lower extremity The characteristic appearance of phlegmasia alba dolens or milk leg is a brawny induration of all tissues and, if ulcerations of the skin are present, findings not unlike those of erysipelas The skin picture is due to lymphedema of the reticular network of lymph vessels These are associated with a chronic *Streptococcus* infection, known in the literature as *lymphangitis streptogenes*

A congenital form of lymphatic obstruction is occasionally seen, known as *Milroy's disease* It may be unilateral or bilateral, and the extremity may attain massive proportions, just as it does in the acquired lymphedema of filariasis

Lymph Nodes

Lymph nodes are accumulations of lymphoid (adenoid) cells embedded in a rich latticework of reticular fibers. They are distributed all over the body along the course of the blood vessels, with important clusters at bifurcations of large vessels. These nodes are filters for the lymph which is brought to them on their way to the thoracic duct. Lymph which traverses a lymph node must pass through its sinuses. One or more such nodes may be traversed by lymph on its way from the tissue fluid to the venous blood Lymph nodes are the site of manufacture of lymphocytes which are formed largely in the germinal centers, where mitotic action is usually seen. Lymph nodes remove particulate matter from the lymph This may include foreign material embedded in an open soft tissue wound foreign material inhaled through the lungs or material injected into the pleural or peritoneal cavities. The lymphocytes manufactured in the lymph nodes are an important source of manufacture of globulins, protein fractions of immunologic importance

Inflammation of a lymph node is referred to as *adenitis* Adenitis may be acute as when draining lymphangitis this is an acute infection, and the adenitis is usually regional to the traumatized or infected area Chronic adenitis may be regional or generalized the chronic infections, such as tuberculosis and syphilis, are usually the causative agents Virus diseases only recently being extensively studied with the aid of electron microscopy are frequently responsible for generalized adenitis. The relationship of virus infections to lymphoid hyperplasia is in the process of elucidation

Tumors of lymph nodes, as of other structures, may be primary or metastatic Metastatic malignancy of lymph nodes is extremely common and is due to transportation of malignant cells via the lymph vessels to their nearest and sometimes even to remote filtering points No further mention will be made of these malignancies here The remainder of the discussion will be devoted to tumors primary in lymphoid tissue

All neoplasia of lymphoid tissue may be grouped under the term *lymphoma* Some pathologists report benign lymphomas such as *giant follicular hyperplasia* (*Brill Symmer s disease*) However other pathologists believe that giant follicular hyperplasia is merely one stage in the development of a malignant lymphoma. Whether all the types of lymphoma are truly neoplastic or not is moot at the present time The possibility that some members of this group are chronic granulomas, initiated by viruses gained some credence in the recent past. Studies in electron microscopy and in cellular chemistry and histology should elucidate this important point. It must be emphasized that malignant lymphomas are not to be considered as neoplasia beginning in one lymph node and metastasizing to others The lymphoma group must be considered as *multicentric* in origin even though all involved nodes may not be found at a given time

Lymphosarcoma is one of the malignant lymphoid neoplasms that arises within lymphoid tissue It may occur anywhere in the body When it becomes blood-borne it is sometimes known as *leukosarcoma* The usual varieties are known as *lymphoblastoma round cell sarcoma* and *reticulum cell sarcoma* Clinically they cannot be differentiated the diagnosis is made by histologic examination Symptoms and signs are produced by infiltration of various organs, which then are symptomatic For example if the masses accumulate in the abdomen, the signs are those referable to the peritoneal cavity if within the mediastinum the symptoms are cardiovascular respiratory

Hodgkin s disease assumes several varieties It affects all ages, particularly the young, and symptoms produced are dependent upon the region involved Histologically the disease is recognized by the disorganization of normal lymphoid structure and the presence of Reed Sternberg cells (a peculiar type of giant cell) All of the lymphomas are accompanied by intermittent fever Histologically three forms of Hodgkin s disease are recognized the *Hodgkin s granuloma* the *paragranuloma* and the *sarcoma*

Treatment of all the lymphomas is unsatisfactory at best Local excision in and of itself rarely suffices to prevent a fatal outcome Irradiation and a variety of chemical cellular poisons may resist the advance of the disease for a while but are not curative

SUMMARY OF PRINCIPLES

1 The anatomy and physiology of veins of the lower extremities are part and parcel of the mechanism of blood return from capillary beds below the level of the heart to the right auricle

2 The venous heart consists of three main components these are the

negative pressure in the pleural space and its changes with respiration the squeezing action of the muscles of the lower extremity and the valves in the veins of the lower extremities

3 Varicose veins are dilated and/or *tortuous veins usually of the super ficial* vessels of the lower extremities Dilatation of the veins usually results in incompetence of their valves

4 The etiology of varicose veins includes two main factors stock, i.e the nature of inherited tissue and chronically increased hydrostatic pressure Clinical examples of chronically raised hydrostatic pressure are preg nancy standing for long periods of time without using the muscles of the lower extremities ascites tumors within the abdominal cavity and constric tive clothing and appliances

5 Superficial saphenous veins which are dilated to accommodate the return blood flow from the lower extremities as a result of occlusion of the deep venous system are known as *secondary varicose veins*

6 *Valvular incompetence* of the saphenous system and of the veins which communicate between the saphenous and the deep systems can be detected by clinical tests.

7 The essential pathologic physiology in varicose veins is ambulatory venous hypertension, a failure of the pressure in the superficial system of veins to drop when the patient exercises the muscles of the legs.

8 Treatment of varicose veins is directed first against the underlying disease if such exists, and then to the veins themselves The stripping operation is more satisfactory than less complete procedures

9 Thromboembolic disease refers to the train of events associated with abnormal intravascular coagulation of blood and to its pathologic sequelae. The predisposing factors are (*a*) stasis of blood (*b*) intimal damage and (*c*) decreased intravascular time *Stasis occurs clinically most often in* (*a*) congestive heart failure, (*b*) any obstruction to the vena cava or the iliac veins (*c*) periphlebitis (*d*) the presence of varicose veins and (*e*) cloth ing or appliance constrictions Intimal damage may be bacterial or mechan ical The endophlebitis is responsible for the adherence of platelets to the damaged intima Later other blood elements are attracted to the roughened surface until a clot is formed Decreased intravascular clotting time occurs clinically in (*a*) hemoconcentration (*b*) polycythemia and (*c*) carcino matosis

10 There are two types of intravascular clots Phlebothrombosis is characterized by red thrombi which are predisposed to most commonly when stasis is the predominating cause. Thrombophlebitis is characterized by *white thrombi in which there is a predominance* of platelets and white cells, and is associated with intimal damage infectious or traumatic Clini cally especially in the later stages these two types of intravascular clots may be found associated with each other

11 Thrombophlebitis is ushered in with both local and systemic signs The local signs are those of infection and a swollen hot and painful ex

tremity the systemic signs are those of fever and leukocytosis Phlebothrombosis unfortunately may be extremely difficult to detect in the ex tremity There may be no local signs whatever or there may be an asymp tomatic increase in the size of the leg Unfortunately the first sign may be the occurrence of a pulmonary embolus

12 The treatment of thromboembolic disease is both prophylactic and direct The prophylactic treatment that is, the prevention of this disease includes the use of anticoagulants where indicated preoperatively the treat ment of infections preoperatively the avoidance of obstructive binders and in general excellent preoperative and postoperative care

13 The definitive treatment of thromboembolic disease once it has occurred includes (*a*) treatment of the infection (*b*) anticoagulants (*c*) relief of pain which is partly due to reflex vasospasm and (*d*) ligation of peripheral veins, especially if a pulmonary embolus has already occurred

14 There are two main complications to thromboembolic disease (*a*) pulmonary embolization the early sequel, (*b*) the postphlebitic syndrome the late sequel

15 Infarction of the lung does not occur with every embolus to the lung Infarction depends upon the size of the embolus and upon the local con ditions within the lung Rapid death after such a catastrophe may follow cerebral signs (hypoxia of the brain) and cardiac signs (cardiac arrest me diated through the vagus nerves)

16 The definitive treatment of pulmonary embolization must be logical and rapidly applied. Atropine should be given to avoid vagal reflexes Pa paverine a vasodilator should be used preferably intravenously Anti coagulants should be started immediately and appropriate venous liga tions must be considered Antibiotics may help avoid pneumonitis in the infarcted area. Pulmonary artery embolectomy is only rarely feasible.

17 The late sequel of thromboembolic disease is known as the *post phlebitic syndrome* It consists of a swollen and painful lower extremity and, ultimately of disabling skin lesions including ulcerations.

18 The genesis of the skin lesions is not completely understood, but probably is associated with a periphlebitic inflammation and resulting fibrosis The resemblance to peptic disease of the duodenum is striking

19 The treatment of the postphlebitic leg is unsatisfactory at best Rest of the extremity with elevation of the leg and antibiotics to control the cellulitis are the usual conservative measures tried first. Operative meas ures include ligation of the deep vessel in which the valves have been de stroyed and excision of the ulcerated and pigmented area with replacement by a split thickness skin graft

20 Phlegmasia cerulea dolens is the spectacular clinical picture seen with sudden massive thrombosis of the femoral or iliac veins of an extremity It may be associated with shock and rapid gangrene of the extremity This situation constitutes an emergency requiring immediate treatment of shock and thrombectomy

21 The lymphatic system consists of endothelium lined vessels which transport fluid from the tissue spaces through filters (lymph nodes) into the venous system

22 Numerous functions of the lymphatic system are known others are not It is a first line of defense against infection from without, particularly with reference to particulate matter Transportation of the largest portion of absorbed fat from the intestinal tract is another major function of the lymphatic system Additionally it acts as a buffer mechanism in rapid adjustments of fluid and electrolytes The contribution of the thoracic duct to the venous flow has variously been estimated at 50 to 120 ml per hour

23 Lymphangitis is the presence of infection in the lymphatic stream There are two types (*a*) reticular or capillary is the presence of infection in the fine meshwork of lymph vessels common in the skin An example is erysipelas an infection with *erysipelatis* an intracutaneous infection (*b*) Tubular lymphangitis is the presence of an infection in the lymphatic chain of vessels from a peripheral tissue to a group of lymph nodes

24 Tumors of lymph vessels are uncommon they are known as *lymphangiomas* Varicosities of lymphatic channels lymphangiectases are probably due to stasis

25 Injuries of the thoracic duct are not uncommon in surgery within or injuries to the chest and peritoneal cavities They are detected by the extravasation of chyle into these body cavities Ligation of the torn duct suffices Collateral channels develop readily

26 The lymphatic channels in the lower extremity may be obstructed by parasites such as filariae, and lead to gigantic swelling of the lower extremity known as *elephantiasis* A congenital form of lymphatic obstruction is occasionally seen known as *Milroy s disease*

SUGGESTED READINGS

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The Total Management of the Surgical Patient The Preoperative, Operative, and Postoperative Periods

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INTRODUCTION

Surgery is a method of treatment of disease employing manual or operative techniques In making use of these methods the surgeon's duties and responsibilities in the management of the patient neither begin nor end with the operative tour de force Rather the total management of the surgical patient begins at varying intervals before the scheduled time of operation, frequently long before the patient is even admitted to the hospital It includes the physical and mental preparation of the patient and the deliberate evaluation of all possible pre-existent and concurrent pathologic states Essential, of course is the restoration to or as near as possible to normal of all known anatomic, physiologic, and psychic derangements The purpose of such extensive preoperative preparation is to render as safe as possible the patient's progress through the operative ordeal and the postoperative recovery phase. The operative assault itself has become more and more extensive in recent years, owing to the widening range of surgical perspectives together with advancement in collateral fields Surgical therapy has further been rendered more safe by a better understanding of the mechanisms of postoperative com

plications together with the physical physiologic, and pharmacologic methods of their treatment.

Little if any new material will be presented in this chapter Much of what has been stated in detail earlier in this book will be coordinated and the relative role of each subject in the total management of the surgical patient will be indicated In effect this chapter constitutes a summary of the Summaries of Principles which apply to all surgical patients, regardless of the specific disease being evaluated and treated

PREOPERATIVE MANAGEMENT

Nutrition and Homeostasis

Success in an elective surgical procedure may be compromised by failure to evaluate the patient s general status. This state of well-being is discussed in terms of nutrition and homeostasis in their broadest meanings They denote a wide conformance to many physiologic norms which reflect a variety of organic functions. These include (1) a normal hemogram whose elements are the red series the white series, platelets and the bleeding-coagulation profile (2) liver profile, including at least some of the major hepatic functions as indicated by the total protein and albumin/globulin ratio serum bilirubin prothrombin time and at least one of the flocculation tests (3) a few basic blood chemical determinations including sugar N P N or urea nitrogen and a serologic test for lues (4) a complete urinalysis, including microscopic study this together with the blood N P N or urea nitrogen is almost always adequate to detect gross renal insufficiency (5) roentgenographic study of the chest which suffices in most cases as a screening mechanism for tuberculosis pulmonary edema and cardiomegaly (6) electrocardiographic study which will occasionally reveal cardiac disease not obvious on physical examination Other studies may be carried out according to hints and suspicions gained by a complete physical examination, which, of course precedes all else in diagnosis and evaluation If any suspicion exists, for example with regard to the fluid and electrolyte balance or to the presence of chronic shock (chronic hypovolemia) these pathologic states may be more carefully defined by added procedures such as the determination of total circulating volume preferably by the radioiodinated serum albumin technic, and a chemical analysis of the blood for the more important electrolytes.

Time spent in elevating the reduced hemoglobin and protein values toward normal and in reconstituting the circulating volume toward its estimated normal value, yields rewarding dividends in the ability of the patient to respond favorably to surgical intervention. Healing will be faster and complications fewer than they would be were these important broad areas of surgical management neglected Specific methods for the correction of hypovolemia, hypoproteinemia and electrolyte imbalance are discussed in detail in preceding chapters.

Pre-existent Disease

Cardiac and Vascular Cardiac evaluation must be made of all patients subjected to operative intervention This may be more casual and routine in the young and healthy person who is usually better able to withstand the surgical ordeal and more detailed and exhaustive in the older patient with known or suspected limitation of functional capacity For elective surgical procedures this medical examination should be as unhurried and extensive as the case warrants For emergency operation cardiac supportive measures must be instituted as quickly as possible and maintained through and after the operative procedure

Thromboembolic disease is an ever present specter which has not diminished in incidence despite all the discussion and attention devoted to it in recent years Prophylaxis of this dread group of complications begins with an awareness of predisposing

factors and meticulous efforts to avoid them The entire subject is treated in detail in Chapter 11 under Thromboembolic Disease Some of the principles will be touched on here for emphasis

Three main factors predispose to the occurrence of thromboembolic phenomena These are (1) *stasis* occurring especially in persons who have been bedridden for long periods of time (cardiac impairments) and who have obstructions of the inferior vena cava and its main tributaries or of the portal circulation (tumors, ascites, pregnancy, etc.) debility and obesity constitute an additional factor in stasis (2) *intimal changes* which may be secondary to inflammation as is seen in pre-existent cutaneous infections (fungus, traumatic, other) in pelvic phlebitis (especially puerperal) in trauma and in intestinal obstruction (3) *rapid intravascular clotting time* as is encountered in polycythemias thrombotic thrombocytopenic purpura, severe dehydration and other disease states (carcinomatosis) The preoperative management of the surgical patient includes a search, by history and physical and laboratory examination for any of the above derangements These measures are detailed in previous chapters and, in general include appropriate antibiotic therapy for infections, correction of cardiac decompensation the attention of the hematologist to altered clotting values and weight reduction for the obese Varicose veins or most particularly pre-existent deep venous phlebitis should be treated either by preoperative elastic compression of the lower extremities in mild cases or by actual operative correction before other surgery is planned The patient is instructed, in the preoperative period that leg exercises deep breathing, and early ambulation are part of the treatment in the postoperative phase of his care

Respiratory Chronic pulmonary disease predisposes to postoperative pulmonary complications Oral, nasopharyngeal, and sinus sepsis is treated before the patient is admitted to the hospital. Chronic pulmonary sepsis (bronchitis and bronchiectasis) is treated vigorously with antibiotics bronchial antispasmodics and expectorants These are discussed in detail elsewhere Particular attention is paid to improving ventilatory function in the patient with emphysema who is most predisposed of all patients to hypoxia and hypercapnia in the postoperative period All of the aforementioned modalities of treatment are employed In addition a positive and negative respirating machine is occasionally useful in avoiding metabolic imbalance due to wide swings in respiratory alkalosis and acidosis. Preoperative bronchoscopic aspiration in these patients is frequently necessary and rewarding

Renal Acute glomerulonephritis and renal insufficiency will preclude other than emergent operative procedures. Renal insufficiency may be relative and must be assessed by the methods described in the section on Nutrition and Homeostasis Azotemia on a renal basis is the concern of the internist and postrenal azotemia the concern of the urologist whereas prerenal azotemia (acute or chronic shock) is the concern of the surgeon He must anticipate it and avoid it or treat it

Metabolic. Obesity is a constant cause for concern to the surgeon Technical difficulties may be greatly multiplied, and hazards in the postoperative period are definitely increased Fat heals poorly wound infections are more common thromboembolic disease is more frequent, and the stress of surgical procedures is, in general less well tolerated. Time taken for a carefully planned weight reduction program is usually rewarded by a much smoother postoperative course

Pre-existent diabetes is a matter of special concern Mild and well-compensated diabetes probably subjects the patient to no added risk. Severe or uncontrolled diabetes on the other hand, renders progress of such a patient through an operative ordeal more hazardous from several points of view In such patients there appears to be an increased incidence of postoperative infections which is incompletely understood as well as a pre

disposition to the associated derangement in fat metabolism atherosclerosis, and its predilection to occlusion of small blood vessels (brain heart, retinas, kidneys and toes) In the preoperative management of the diabetic patient attention must be paid particularly to the prevention of metabolic organic acidosis (ketosis) as well as the maintenance of a stable near normal blood sugar level. Insulin, type and amount, is used as necessary The spillage of a small amount of sugar in the urine is of much less importance than the presence of ketonuria.

The state of adrenal sufficiency is a matter of concern to the surgeon today This is because of the large number of patients who have received or are still taking, steroid preparations, which are probably overused. The added stress of the surgical assault must be compensated for by the continuation of and even increase of administered steroids This is particularly true since preoperative administration of the steroids may have suppressed the endogenous hormonal secretion and the ability to respond to induced stress. Knowledge of adrenal disease is likewise of great importance to the surgeon because armed with this information he can carry such patients through surgery with adequate support, more successfully than ever before Surgical procedures in general are not undertaken lightly in such patients, since metabolic deficits in true Addison s disease are as great as any encountered in medicine.

Hepatic disease may be overt or latent. Obvious hepatic insufficiency will preclude all but emergent procedures Patients with milder forms of this disease will do poorly postoperatively and may be thrown into acute hepatic failure unless all precautionary supportive measures are taken (and even sometimes despite them) Reference is made to the section on Nutrition and Homeostasis Supportive therapy includes high carbohydrate intake parenterally correction of hypoproteinemia and hypoprothrombinemia with parenteral synthetic vitamin K and large doses of the vitamin B complex. Liver biopsy by the percutaneous needle technic may aid in the preparation of the patient by pinpointing the diagnosis which usually falls into three main categories Laennec s type of cirrhosis, virus-caused hepatic cirrhosis (postnecrotic) and metastatic malignant disease Hepatic failure seems to be associated with the development of ammonia intoxication. Glutamic acid has been of value in combatting this metabolic derangement.

Extremes of Age Special problems pertaining to children and to the aged constitute the subject matter of entire chapters and even books. Only the salient features in the management of these two special groups will be mentioned here Infants and children have the recuperative facilities that go with the most intensive growth period and highest metabolic activity of the entire human life span. Nonetheless the infant and child are not just small adult human beings, but have special problems associated with the increased ratio of body surface to body weight and the greater importance of a relatively small blood loss in producing hypovolemia Careful estimate of total body fluids proteins and hemoglobin with restoration of these values to normal preoperatively is rewarded by improved postoperative results Care of the aged patient is an everyday problem in modern surgery since more than 11 per cent of the total population of the United States is now over the age of sixty five this figure will rise appreciably during the next generation The degenerative processes in tissue that are a part of life make operative intervention in general more hazardous and more likely to be associated with complications They demand a fine focus of attention by the surgeon in the preoperative period. There are no new principles for the elderly patient merely a greater emphasis on those which apply to all All of the factors outlined in the section on Nutrition and Homeostasis must be part of the check list in the preparation of those in the twilight of life If the risk of operative procedure in any given elderly patient is believed to exceed that of the disease he manifests it is wiser not to operate Particularly is this true in the case

of pulmonary insufficiency severe myocardial insufficiency, and hepatic or renal failure

Psychiatric The problems of mental disease must also be considered Psychiatric disturbances may effectively preclude successful rehabilitation by a surgical procedure Some such patients may even have their mental illness aggravated by the trauma of surgery In addition to extending every kindness to such patients and helping them understand the problems that face them in the future the surgeon must make use of the help and advice of the trained psychiatrist.

Immediate Preoperative Care

When all pertinent data have been collected and evaluated and when all corrective measures for discoverable derangements have been carried out, the operation is scheduled with the knowledge and consent of the patient If the patient is mentally incompetent a responsible member of the family is consulted in his stead For the intelligent patient a review of the illness and the proposed operation is usually appreciated and will make the patient more cooperative A similar visit by the anesthesiologist and an explanation of procedure to be carried out in the operating room is of great help in allaying the patient s apprehension Emphasizing the extreme importance of deep breathing to avoid pulmonary atelectasis and of movement of the legs in bed to avoid stasis in the postoperative period is very helpful at this time in attaining a smoother postoperative course Appropriate cutaneous areas are shaved and prepared by washing the night before operation. An enema is given (unless there is a specific contraindication) the night before operation in order to empty the lower bowel this obviates undesirable reflexes on the operating table and makes the early postoperative period more comfortable Sleep medication is used as necessary In the morning bladder catheters are placed, stomach tubes inserted, and special intravenous medication started before the patent leaves the floor Preoperative medication is designed to reduce to a minimum the natural and understandable fear and apprehension to which a patient is subjected in the minutes before consciousness is lost Movement from bed to cart and cart to operating table should be as gentle as possible In special situations, such as the presence of unreduced bone fragments and in thyrotoxicosis it is frequently wise to induce anesthesia before the patient is moved to the operating room.

The Emergency Case

In the preoperative preparation of the emergency case one may not have time to go through the entire check list detailed in the above two sections *Timing* of surgical intervention in such situations is of the greatest importance One must take time minutes to a few hours, necessary to correct those factors which may preclude the successful outcome of the operation Only the broad areas of preoperative management will be indicated here The details are listed in the chapters in which those diseases and situations which so frequently give rise to emergencies are discussed They are in general intestinal obstruction, severe burns exsanguinating hemorrhage from certain diseases and severe trauma.

Unless time is taken for the rapid control of those derangements which would terminate life before the patient is even brought to the operating room no surgical procedure can be contemplated These dire circumstances include the control of exsanguinating hemorrhage by whatever means possible short of entering major body cavities, the immediate relief of *asphyxia* usually by emergency tracheostomy and the correction of *shock* by replacement of lost circulating volume preferably by blood but by plasma or plasma expanders or even water-electrolyte solutions until blood is available. These procedures command priority over all other considerations Decompression of the upper gastrointestinal tract by appropriate intubation when the diagnosis of mechanical intestinal obstruction has been made is mandatory and frequently lifesaving If acute

gastric dilatation is detected or vomiting is in progress when the patient is first seen or the patient has recently eaten the stomach must be emptied by aspiration as thoroughly as possible and, preferably a tube allowed to remain in the stomach Continued vomiting in the unconscious state or as the patient is being anesthetized may result in the aspiration of gastric contents into the tracheobronchial tree, with the resultant aspiration pneumonitis, which in the aged and debilitated patient may be the difference between life and death

OPERATIVE PERIOD

The operation is the climax of surgical care Its success depends upon the nature and severity of the pathology the ability of the patient s general body economy to withstand the operative trauma, and to no small extent, upon the technical skill, judgment, and experience of the surgeon. The fact that this volume deals with the principles involved in surgical diagnosis and therapy in no way minimizes the extreme importance of technical training of the surgeon including a knowledge of surgical pathology and technical dexterity The details of these considerations do not fall within the scope of this book.

Anesthesia

Modern surgery s recent advances are due in no small measure to collateral advances made in the field of *anesthesiology* Extensive surgical ventures are made possible by cooperation between the surgeon and anesthesiologist. The anesthesiologist of today is a highly skilled and thoroughly trained medical specialist who is completely conversant with cardiorespiratory physiology the use of blood and substitutes to balance blood lost by disease and during operation and the science and art of the use of drugs before and during the time of anesthesia. Both he and the surgeon have an appreciation of all the factors involved in the total body response to the surgical ordeal

Drugs used as preoperative medication are usually from one of the following groups (1) *narcotics* to allay apprehension and diminish response to painful stimuli (these drugs also lower oxygen consumption) (2) *barbiturates* for sedation and diminution of any adverse response to drugs which may be used subsequently (3) *parasympatholytic drugs* to reduce the likelihood of reflexes which may be mediated through the vagus, and thus lead to slowing of the heart, and to depress the secretions of the upper respiratory tract. *Antihistaminic drugs* have been used in recent years partially for their sedative effect and partially to counteract sensitivities and idiosyncrasies to drugs and blood administered while the patient is under anesthesia They reduce the amount of both narcotics and anesthetic agents needed during and immediately after the operative procedure

The anesthesiologist is trained to decide which agents are safest for a given patient, as determined by the nature of the patient s illness and the anesthesiologist s familiarity with a particular technic and drug For example the anesthesiologist will rarely use intravenous Pentothal as the main medication if the patient is in a relative state of shock and will certainly desire to use such agents as make the employment of the highest concentration of oxygen possible when hypoxia already exists and when there is pre-existent cardiac or respiratory disease

Spinal anesthesia will usually be his choice in pre-existent disease of the respiratory tract unless the operation is within the chest itself The anesthesiologist will visit the patient the day prior to operation and familiarize himself with the salient features of the disease and the proposed operation his explanation of any anticipated procedures to the patient usually allays the patient s apprehension and makes him more cooperative This is particularly true when dealing with children

ing to the indications for this procedure and the experience of the anesthesiologist. In good hands maximal muscular relaxation is obtained with a minimum of postspinal head aches especially when the very smallest of needles is used.

Many surgical procedures can still be carried out successfully with a minimum of postoperative discomfort by local nerve block and/or regional infiltration with local anesthetics The patient must be cooperative and the ability of the surgeon to accomplish his task must not be hindered by the anesthetic technic. Although even major surgical procedures can be carried out under regional infiltration the technic should be carefully chosen only when it suits the circumstances including the psychic make up of the patient *Topical anesthesia* for endoscopy is properly included under the heading of local anesthesia Adverse reactions to the use of local anesthetic agents can be minimized by the preoperative use of barbiturates and antihistaminics 1 hr or more before the local anesthesia is induced One must avoid too high a concentration of the drug in the blood stream at any one time Oxygen and a mask a vasopressor drug antihistaminics and barbiturates should be available in the room whenever local anesthesia is employed

Cardiorespiratory Failure

Cardiorespiratory failure more commonly referred to as cardiac arrest, occurs in young and healthy patients as well as in debilitated very ill poor risk patients This operating room catastrophe is discussed in detail in Chapter 20 to which the reader is referred. Suffice it to say here that the principal cause of cardiorespiratory failure is *hypoxia* either pre-existent or induced in the operating room The lack of proper oxygenation may be a part of the patient s disease (emphysema chronic suppurative pulmonary disease chronic coronary insufficiency pneumonitis ventilatory insufficiency induced by trauma to the chest, or elevated diaphragm due to intraabdominal disease) *Reflexes* due to noxious stimuli or to traction on viscera mediated through the vagus nerve, are more serious in the presence of hypoxia than under conditions of adequate oxygenation The use of atropine or atropine-like drugs in the preoperative medication schedule is of great aid in avoiding this catastrophe It should be pointed out that the human being is a markedly vagotonic animal and can tolerate large doses of atropine Without this medication bradycardia induced by vagus-mediated reflexes renders the heart more susceptible to arrest Hypoxia, even of a few seconds duration, must be avoided by the anesthesiologist during induction. Particularly important is slow induction in cardiac patients, so that no laryngospasm or even temporary reduction in oxygenation occurs.

Cardiac arrest means that there is an *ineffective cardiac beat* This situation obtains in either of two types of response of the heart to the instigating hypoxia. The first of these is *asystole* in which the ventricles are found to be flaccid and not beating at all the other is *ventricular fibrillation* in which the musculature of the ventricles is twitching in an uncoordinated and ineffective manner The result of either type of pathology is the *reduction of cardiac output* to zero With no blood being put out from the left ventricle no oxygen can be carried to the brain, and in about four minutes the respiratory center in the medulla will cease to function thus the term *cardiorespiratory failure*

As outlined in Chapter 20 cardiac arrest is treated by immediate opening of the left chest cavity while the anesthetist administers 100 per cent oxygen by positive pressure through an endotracheal tube When the chest and the pericardium are opened, the heart is observed to see whether it is in fibrillation or standstill Under any condition intermittent compression of the heart (*manual systole*) is immediately instituted this procedure has been erroneously referred to as cardiac massage With manual systole being sustained at a rate of 40 to 60 per minute and oxygen being administered, more deliberate plans can be made for definitive restoration The method and drugs used for

restarting the heart in case of asystole are discussed in Chapter 20 If ventricular fibrillation is observed the heart may be defibrillated and the resultant asystole treated as it would have been had it occurred primarily Every surgeon regardless of subspecialty should be familiar with the diagnosis and treatment of cardiorespiratory failure and should be prepared to institute restorative measures. The anesthesiologist is an integral part of the operating team and a vital part in the resuscitation of such patients and frequently will be the first to detect the presence of this dreaded complication There should be liaison between surgeon and anesthesiologist at all times with regard to the location of equipment necessary drugs available and procedure to be followed should this complication occur It is predicted that before too long monitoring devices will be attached to every patient who is under anesthesia, in order that a visual and/or audible signal of each heartbeat may be seen or heard by the anesthesiologist

Recovery and Intensive Therapy Room

The modern operating suite is complemented by a department which receives the patient from the operating room and which provides intensive care until that patient is conscious and able to respond to his attendants In the recovery room will be found all facilities for resuscitation from the state of shock (blood and blood substitutes) and ventilatory insufficiency (automatic respiratory machines) and equipment for aspirating the tracheobronchial tree Nurses carefully trained in the care of those emerging from anesthesia are in attendance The recovery room, all of its apparatus and personnel, are under the direct supervision of the department of anesthesia. The patient leaves the recovery and intensive therapy room by permission of the anesthesiologist when he considers floor care adequate His trained eye is helpful in differentiating between hypovolemic and posthypercapnic shock. He will give particular attention to the avoidance of respiratory alkalosis or acidosis Particular attention is paid to the care of drainage bottles and tubes The recovery and intensive therapy room is considered a part of the operative suite.

POSTOPERATIVE MANAGEMENT

Nutrition and Homeostasis: Total Body Response to Surgery

A discussion of the patient s needs in the postoperative period not only concerns those factors elaborated in detail in Chapter 5 (including maintenance requirements of fluids and electrolytes and restoration of those fluids and electrolytes lost as a result of the surgical procedure or drainage plus the body requirements for tissue repair by proteins and energy requirements by carbohydrates and fats, in addition to vitamins) but must also consider the total body response to the stress of both the illness and the operative trauma. Tissue trauma, accidentally inflicted or occurring during purposeful surgical attack excites a response on the part of the organism which is designed to help it withstand the injury The mechanisms whereby these ends are mediated and effected are not completely understood It is known, however that the inability properly to respond to surgical trauma may preclude recovery from a surgical attack that is well conceived and prepared for and competently executed. A period of anesthesia itself especially if deep or prolonged, contributes to the need for this response.

Without being overly teleologic, it may be said that the automatic and inherent mechanisms in the living body tend to maintain a relatively stable balance of all important body functions and dynamics These are mediated through the reciprocal functions of the autonomic nervous system (see Chapter 9) and through certain humoral mechanisms. The autonomic nervous system is particularly involved in the neurologic control of vascular beds, resulting in a redistribution of blood from less important to more important areas under conditions of stress This concept of

the dynamic control of circulating volume has been called *hematometakinesia* The most important humoral mechanism is mediated through the master endocrine gland the *adrenal cortex*

The response of the adrenal cortex to surgical trauma resembles closely the effect produced by the administration of the hormones of this gland These include pressor phenomena, the mobilization of sugar from the liver the retention of sodium and with it, of water the excretion of potassium and other effects as yet incompletely understood. Evidence of increased activity of the adrenal cortex in the early postoperative period may be detected by the increased excretion of steroids in the urine the reduction in number of eosinophils and other specialized tests There are two conditions under which the adrenal cortex may be incapable of a satisfactory response following major operative procedures and these must be understood and appreciated in advance The first of these obtains when the patient has been subjected to a long siege of illness and debility immediately preceding the operation these stressful days, weeks, and possibly months may have exhausted the ability of the adrenal cortex further to respond to superimposed surgical trauma The second special situation is that in which the patient has been receiving corticosteroid medication over a relatively long period of time Under such conditions the exogenous steroids will suppress the ability of the patient s own adrenals *to respond to a stressful stimulus* (*This suppression of an endocrine substance by the* administration of that substance therapeutically is true of all of the endocrine glands.) Such patients must be supported during and after the operation by the parenteral administration of the adrenal corticosteroids in sufficient amount to maintain normal hemodynamic homeostasis. When parenteral steroid support is to be removed the adrenal glands must be stimulated to renewed activity by administration of adrenocorticotrophic hormone (ACTH) The adrenal cortex may be so damaged that even response to ACTH is inadequate and steroid support must be maintained over long periods of time

The early postoperative period is further characterized by increased tissue catabolism. This is reflected in the increased urinary excretion of nitrogen The increased catabolic rate is a part of the response to stress and possibly may be mediated through increased thyroid hormone secretion Androgens administered during this time may reverse this process and cause the nitrogen to be saved for tissue reconstruction

The maintenance of proper nutrition in the postoperative phase, and particularly in patients in whom the gastrointestinal route is not available is discussed in detail in Chapter 5 There the technic is detailed for the administration of fluids and electrolytes for maintenance and of "repair" fluids for abnormal losses as from spontaneous or surgical fistulas and from decompression tubes The optimal use of proteins during the post operative period is based on certain facts which are worthy of repetition Protein is most useful to the body when it is ingested orally Amino acids administered parenterally are expensive and inefficient. Furthermore the administered amino acids will be broken down and used for energy purposes unless carbohydrates in large amounts are given simultaneously The necessity of supplying potassium ion to facilitate transit of glucose into the cell is re-emphasized The most satisfactory method of maintaining protein norms by the parenteral route is the *administration of blood and plasma* Since hypoproteinemia is almost universally associated with hypohemoglobinemia, whole blood is found most useful

Hypovolemia, induced during the operation or antedating the operation may still be present in the postoperative period and may preclude or delay normal recovery and healing. The hemogram alone as an indication of the total circulating mass of hemoglobin may be grossly misleading Determination of the total circulating volume preferably by the radioiodinated serum albumin technic, together with the hematocrit and hemoglobin

Thrombophlebitis is characterized clinically by evidence of venous obstruction and by inflammation the signs of inflammation are both local and generalized If the thrombophlebitis is extensive, the associated arterial spasm mediated through a low cord reflex is prominent. Phlebothrombosis, unfortunately more often than not is silent until a fragment of the thrombus has broken off and become a pulmonary embolus It may, however be suspected and occasionally detected by the asymptomatic swelling of a calf and occasionally by the isolated finding of a tachycardia unrelated to a temperature rise It should be pointed out that frequently by the time the clinical diagnosis of thromboembolic disease is made the two processes may coexist even though the original process was clearly one or the other Thus, a thrombophlebitic clot which is largely adherent to the vein may be loose at its central end (tail part) with a tendency to break off and become an embolus.

Despite awareness of thromboembolic disease and zeal in avoiding it and its sequelae in recent years, this ideal is difficult of accomplishment. The avoidance of these accidents involves a series of measures which begins before and continues during and after the operation The preoperative measures include the eradication of local infection the treatment of pre-existent varicose veins and/or the postphlebitic syndrome (a sequel of previous deep venous occlusion) the preoperative bandaging of legs in patients with varices or previous deep venous occlusion and the maintenance of a normal circulating volume with a balanced water and electrolyte pattern During the operation care is taken that there is no undue pressure on the calves or the heels and that the legs do not overhang the edge of the operating table without adequate cushioning The avoidance of hypotension during the operative period is also helpful In the postoperative period the external compression of the lower extremities, especially in the case of pre-existent varices and deep venous occlusion is continued deep breathing exercises are encouraged to aid in emptying of the veins toward the right heart and, particularly leg exercises are encouraged which involve active motion of the calf muscles The use of restricting abdominal binders is interdicted *Early ambulation* has as one of its main goals the effective use of the lower extremity muscles in massaging the venous return toward the heart It is uniformly employed even in major cases unless a specific contraindication exists When there is a history of previous thromboembolic disease prophylactic anticoagulant therapy may be used with discretion according to the nature of the operative procedure and hematologic findings.

The active treatment of thrombophlebitis is the treatment of an intravascular clot and an infection In superficial thrombophlebitis pulmonary embolization rarely occurs The treatment consists essentially of the use of antibiotics rest, immobilization and elevation of the lower extremity and hot, moist packs for a reactive hyperemia. Anticoagulants are used so that the process will not be extended proximally

The active treatment of phlebothrombosis, if the diagnosis is made before a pulmonary embolization occurs may take one of two pathways. The first involves essentially the use of anticoagulants to render the circulating blood effectively uncoagulable This is accomplished most rapidly with the use of heparin which may be administered intravenously or intramuscularly with immediate action Antiprothrombin drugs may then be resorted to which may be taken orally and are less expensive their action is more easily followed. When they are employed a daily prothrombin level determination is necessary and the anticoagulant for the day is not ordered until the morning prothrombin level is reported The acceptable effective level is a prothrombin range of 20 to 40 per cent of normal The alternate acceptable method in the active treatment of phlebothrombosis of the lower extremities is the exploration and ligation of the (so-called) superficial femoral vein The vein is opened any clot available to aspiration is removed and the vein is

ligated in continuity so that no clot may be propagated toward the lungs This procedure is almost always done bilaterally even though the phlebothrombosis occurs in one leg since altogether too often after ligation on what was thought to be the diseased side a fatal pulmonary embolus may occur from the opposite side Results reported from many groups throughout the country indicate equally good results with either anticoagulation therapy alone or venous ligation

The dreaded sequel to phlebothrombosis is *pulmonary embolus* the movement of a venous thrombus to the pulmonary circuit. Unfortunately the first inkling that a phlebothrombosis of the lower extremity exists may be the catastrophic occurrence of a pulmonary embolus This occurs postoperatively in clinically detectable form in about one out of each one thousand major cases, with death in about one out of each five thousand cases Infarction of the lung does not always follow the lodging of an embolus in a pulmonary artery Owing to the double circulation of the lung, viability is frequently preserved provided passive congestion does not exist. Massive pulmonary emboli are those which occlude an entire pulmonary artery or its primary division Such catastrophes are heralded by a sudden pain in the chest, gasping respiration bradycardia and syncope and occasionally cardiac arrest and sudden death. The development of the signs of cor pulmonale on the electrocardiogram is corroborative evidence Many small showers of pulmonary emboli may occur with minimal symptoms and may precede a massive embolization When infarction occurs there may be a cough associated with hemoptysis severe pain due to pleuritis and after 2 or 3 days roentgenographic evidence of an atelectatic pulmonary segment Death from embolism is due not only to the arterial obstruction but to reflexes to the heart via the vagus which induces bradycardia and possibly arrest spasm of the vessels of the peripheral pulmonary arterial tree may also result These provide the indications for therapy

The active treatment of pulmonary embolization is one of emergent character Oxygen therapy should be instituted as soon as possible Atropine should be administered preferably intravenously for the first dose in relatively large doses in order to avoid or minimize the pulmonocardiac reflexes mediated through the vagus nerve The simultaneous administration of papaverine also preferably given intravenously may help to propagate the pulmonary embolus distally that is into as small a vessel as possible Anticoagulant drugs should be started immediately Bronchodilators may be very helpful in relieving the asphyxial sensation

Once a patient has suffered a recognizable but nonfatal embolism the underlying phlebothrombosis should be treated by venous ligation rather than anticoagulants alone The choice and level of vein to be ligated have been discussed in Chapter 11

Respiratory Respiratory complications following surgery are the result of either or both of the following (1) pre-existent respiratory infections that have been inadequately treated (2) ventilatory dysfunction and insufficiency leading to postoperative atelectasis and its sequelae In all patients subjected to elective operations the respiratory system should be thoroughly examined and evaluated and any infections treated This includes the eradication as far as possible of chronic sinusitis, sinobronchial disease, chronic bronchitis and chronic suppurative pulmonary disease (bronchiectasis) Treatment of these pre-existing conditions is outlined in appropriate preceding chapters and includes eradication of foci of infection, antibiotic therapy inhalation bronchodilators and wetting agents and where necessary bronchoscopy

The most common cause of postoperative pulmonary complications is *inadequate ventilatory excursion* in the early postoperative period Shallow breathing is the direct result of pain which is experienced on deep inspiration and the sequel to deep sedation Operations within the thoracic cavity and the upper portion of the abdomen are particularly

prone to result in limited ventilatory excursion Similarly limitation of diaphragmatic motion reduces the respiratory depth The patient pants rather than breathes

Inadequate respiratory excursion allows *the accumulation of bronchial secretions*, particularly in the very small bronchioles The air is absorbed from their alveoli and they become atelectatic Bronchial secretions are not expelled, because the tussive effort is markedly diminished, since coughing further produces pain The real pathogenesis, then, of postoperative atelectasis is not primarily *the bronchial secretions but the failure to breathe deeply* owing to the causes enumerated above The patient should be told about this in the preoperative period and asked to breathe deeply after the operation even though it may cause him some pain Co-operation is much easier to obtain when the entire subject is presented in its proper light *beforehand It will be recalled that deep* breathing is also essential in avoiding venous stasis of the lower extremities (see Chapter 11)

Pain on deep breathing is best obviated by local anesthetic injection into the appropriate intercostal nerves This procedure permits normal ventilatory function without oversedation The inhalation of carbon dioxide either from a tank of this substance (or Carbogen, 93 per cent oxygen and 7 per cent carbon dioxide) or by means of rebreathing from a closed bag is of adjunctive aid in producing an increased depth of inspiration Early ambulation aids in deep breathing and in increasing tussive effort, resulting in the expulsion of retained bronchial secretions An apparatus known as the *exsufflator* applies negative pressure of 30 to 40 cm H_2O to the patient's respiratory tree via a tight fitting face mask and is frequently very helpful in dislodging thick secretions which cannot be coughed up Tracheobronchial toilet and bronchoscopy are occasionally necessary these measures are discussed in appropriate chapters Tight dressings of the upper abdomen and of the thoracic cage limit respiratory excursion and are to be discouraged When any or all of these measures are employed as is appropriate in each case postoperative atelectasis will be reduced to a minimum and with it the incidence of pneumonitis

Atelectasis and pneumonitis are recognized in the postoperative period by a rise in temperature not otherwise explained a short hacking cough dullness in a given area of the lung, and later x ray evidence of an airless segment or lobe Treatment of this complication is discussed in Chapter 16 however meticulous attention to the above mentioned details will largely eliminate the necessity for such treatment.

Renal and Lower Urinary The most common postoperative complication of the kidneys is acute renal insufficiency As detailed in Chapter 5 there are three types of renal insufficiency (1) renal, (2) prerenal and (3) postrenal The renal causes refer to *glomerulonephritis When this disease exists*, no surgical procedure will be performed except for urgent necessity

The surgeon is most concerned with the *prerenal* forms of azotemia The cause of this form is essentially prolonged shock i e., a period in which there is a reduction in the circulating blood volume and hypotension This pathophysiologic state occurs, as a rule as a result of hemorrhage or of the severe dehydration and electrolyte depletion occurring in intestinal obstruction or extensive burns Renal shutdown is brought about by a profound reduction in glomerular filtration pressure The renal shutdown may be partial (oliguria) or complete (anuria) For a detailed discussion of the three phases of acute anuria and/or oliguria and the treatment of each, the reader is referred to Chapter 5

Lower nephron nephrosis is a term applied to a type of renal insufficiency in which the pathologic process is thought to be in the distal convoluted renal tubules and in the proximal portion of the loop of Henle These changes occur as a result of the transfusion of incompatible blood or the administration of toxic drugs or following massive trauma (the crush syndrome) In all there is thought

to be obstruction of these tubules by pigments from hemolyzed erythrocytes (hemoglobin) or from destroyed muscles (myoglobin)

Urinary retention or the inability to empty the bladder is a frequent occurrence in the early postoperative period It occurs especially in pelvic surgery where the innervation of the bladder may be interfered with and when operative pain prevents straining Additionally urinary retention is produced by the excessive administration of narcotics which cause a contraction of the sphincter at the outlet of the bladder Cholinergic drugs may be administered parenterally to cause a contraction of bladder muscle providing such drugs are not contraindicated by pre-existent or concurrent cardiac respiratory or intestinal disease If repeated catheterization is necessary it is preferable that an indwelling catheter be placed to lessen the chances of infection of the bladder (cystitis) with a possible ascending urinary tract infection When repeated catheterization or an indwelling catheter is necessary appropriate antibiotics should be used. It is important to recognize that frequent urination of small amounts may be an *overflow phenomenon* of a distended bladder Catheterization for residual urine will ascertain this fact, and an indwelling catheter should be allowed to remain in the bladder until the musculature regains its tonus or the neurologic disease which underlies it has been overcome

Gastrointestinal Nausea and vomiting are commonly encountered in the early postoperative period and are usually due to the inhalation anesthetic agent and to mechanical trauma especially in the upper abdomen Reverse peristalsis and, with it, the nausea and vomiting usually subside in a few hours. Antiemetic medications are available for parenteral administration

Adynamic ileus with ensuing abdominal distention is the rule rather than the exception after major abdominal intervention. It is due essentially to the handling of the peritoneal viscera but may be aided and abetted by the presence of shock and electrolyte imbalance particularly hypopotassemia Time and intestinal tube decompression from above are curative One must repeatedly reevaluate the occurrence of ileus which persists in the postoperative period, however for occasionally a mechanical intestinal obstruction is created by the operative procedure This will of course demand early reoperation

Acute gastric dilatation is an ileus of the stomach which may be a part of the general picture of ileus but which occasionally goes on to such proportions as to threaten life Mechanical obstruction of the pyloric outlet or the duodenum or high in the jejunum must be suspected Acute gastric dilatation is accompanied by a shocklike picture associated with belching and retching· singultus (hiccups) may be prominent The vomitus is observed to be either dark green or black in character The stomach should immediately be intubated, and as a rule a very large amount of gastric material will be aspirated (500 to 2,500 cc) Gastric suction must be continued until peristalsis is normal

Postoperative *singultus* (hiccups) may also be caused by inflammatory or chemical irritation of the diaphragmatic peritoneum by other than acute gastric dilatation. Other conditions to be considered are intestinal obstruction purulent peritonitis diffuse fat necrosis, as is found in acute pancreatitis uremia and carcinomatosis peritonei. Treatment is usually ineffective unless the cause is found

The Wound Postoperative complications in the healing of the surgical wound include infections superficial dehiscence and evisceration *Wound infections* are usually a result of the direct contamination of the wound by pathogenic bacteria, either introduced at the time of surgical therapy by the operating team or introduced from an open viscus, as in emergency bowel resection without preparation of the intestinal tract. *Hemorrhage* within the wound, a result of either inaccurate hemostasis at the time of operation or an abnormal bleeding-coagulation profile is

conducive to the formation of a hematoma, which is then susceptible to infection

Superficial *wound dehiscence* is usually a sequel to superficial infection. *Evisceration* of a wound implies complete lack of healing of cut surfaces or a disruption of the wound after healing may have been expected The factors responsible for poor wound healing are discussed in detail in Chapter 3 In general there are local and systemic causes. The local factors include poor approximation of tissues interposition of fat, strangulation of tissue with ligatures that are too tight, and poor hemostasis The systemic factors which make for poor wound healing, and therefore disruption are hypoproteinemia and hypohemoglobinemia fluid and electrolyte imbalance hypovitaminosis especially of vitamin C and the presence of malignant tissue within the wound Undue straining in the postoperative period may cause early wound disruption The most common causes of such strain are chronic cough severe retching mental disorientation and struggling and prostatism without adequate urinary drainage

Miscellaneous Decubitus ulcers (bed sores) are necrotic areas of skin they are not often seen in the healthy patient who can be mobilized in and out of bed They are most common in the aged cachectic and debilitated patients in those who are totally bedridden and in the patient whose skin is anesthetic, such as the paraplegic and the patient with cord or peripheral nerve injuries The most common sites of decubitus ulcers are over the bony prominences of the sacrum the iliac spines, the ischial tuberosities, the scapulas the elbows, and the heels Bed sores are avoided by early mobilization frequent moving from side to side in bed frequent changes of bed linen especially when soiled or moistened by the incontinent or unconscious patient and meticulous nursing care of the skin Decubitus ulcers due to ill-fitting prostheses and appliances are mentioned only in passing

Extensive decubitus ulcers may have to be treated by wide debridement and closure by skin flaps or free grafts How much can and should be done will depend on the nature of the primary illness and the ability of the patient to withstand further surgical trauma

Acute parotitis is seen much less frequently now than in the preantibiotic period However, it still occurs in the debilitated, aged and cachectic patient Poor oral hygiene and fluid-electrolyte imbalance are predisposing factors Pathogenic organisms enter Stensen s duct in the mouth and suppuration of the parotid gland may follow Treatment of this complication is by both local and systemic means, as well as by correction of the underlying oral sepsis and systemic debility

Psychiatric. Psychoses of various types are a constant source of worry to the surgeon for they frequently preclude a successful rehabilitation of a patient who has undergone a technically excellent operative procedure Frequently the surgeon will be able to detect the likelihood of this postoperative complication in his initial interview with the patient from an analysis of the patient s life history and presenting complaints If there is any doubt, psychiatric advice should be sought before the patient is scheduled for surgery In any event such advice and help may be necessary in the postoperative period especially if the operative procedure was performed as an emergency Chronic alcoholism is probably the most frequently encountered basis of these untoward postsurgical sequelae but adult maladjustments of other types are not at all uncommon

SUMMARY OF PRINCIPLES

No numbered Summary of Principles is appended to this chapter because it is in itself a summary of the principles in all chapters on the total management of the surgical patient

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13

The Thyroid

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BASIC CONSIDERATIONS

Anatomy

The thyroid is the largest single gland of internal secretion (ductless endocrine) Its average weight in the adult varies from 30 to 60 Gm and it is located on either side of the upper trachea and lower larynx straddling these structures ventrally by an isthmus It is encased within the layers of the deep cervical fascia (pretracheal) which constitute its surgical capsule It derives its arterial blood supply from two sources, the inferior thyroid artery from the thyrocervical branch of the subclavian and the superior thyroid artery from the external carotid an occasional thyroidea ima artery arises from the great vessels in the superior mediastinum.

The basic histologic architecture of the thyroid is the lobule, which is composed of numerous acini or follicles, which are lined by a single layer of cuboidal epithelium and which contain a varying amount of gelatinous pink-staining material called *colloid* The morphology of the gland varies with the degree of functional activity In the hypoactive state the epithelium is low cuboidal and the acinar lumen is densely packed with colloid In the active state the cuboidal epithelium is relatively higher and the colloid thinner and somewhat vacuolated Hyper

activity is characterized by hyperplasia with deep-staining cells and scant colloid Further hyperplastic changes include the formation of papillary folds and an increase in the number of acini Blood vessels in the thyroid gland are abundant the thyroid being second only to the adrenal in blood flow per unit weight of tissue per unit of time (about 5 ml/Gm/min) The thyroid gland is similarly richly endowed with lymphatics The nerve supply to the thyroid is from the sympathetic trunks and ganglia in the cervical and upper thoracic divisions.

Physiology and Pathology

The function of the thyroid gland is the regulation of the rate of body metabolism This is mediated through its specific hormone *thyroxin* a globulin containing iodine and the amino acid *tyrosine* The thyroid gland is unique in that it has the capacity to extract ingested iodine from the circulating blood and store it in an organic state The elemental iodine combined with amino acids, is stored as thyroglobulin but secreted as thyroxin Recent evidence indicates that the active metabolic compound is triiodothyronine (T3)

The physiologic action of the thyroid gland was originally studied by pharmacologic methods, i.e. the production of symptoms by the experimental administration of supraphysiologic amounts and, conversely by the ablation of the glands for the total removal of the active principle The effect of administration of the thyroid principle is catalytic, increasing the metabolism of practically all cells of the body and, in so doing increasing the consumption of oxygen and simultaneously increasing the rate of destruction of tissue (*Metabolism* refers to the sum total of all chemical processes and changes whereby the biologic function of living tissue is effected It consists in *anabolism* the constructive or assimilative phase and *catabolism* the destructive or degenerative phase)

The thyroid gland is not self-contained and autonomous it is part of a complex interglandular relationship with other endocrines particularly the anterior pituitary The anterior pituitary gland (hypophysis), among its other functions elaborates a substance which stimulates the thyroid gland to activity this material is known as the *thyrotropic hormone* (TSH) It, in turn is inhibited by excessive formation or administration of thyroxin

A grossly deficient iodine intake may result in the inadequate formation of thyroxin this disease is discussed later in the chapter in detail An excess of iodine administration in a patient with a very active thyroid gland will prevent the further formation of the specific hormone for a limited period of time Numerous drugs, particularly those of the thiourea group prevent the formation of thyroxin within the gland they are known as *antithyroid substances*

Methods of Study

Clinical study of thyroid function includes the following determinations (1) The basal metabolic rate (BMR) measures the rate of oxygen consumption per unit of body surface per unit of time in the basal, or vegetative state Since the essential function of the thyroid gland is the regulation of the body metabolism this test is the most direct measurement of the thyroid status There are however many sources of error in the determination of the BMR, which must be taken into consideration in evaluating its results Almost all the errors lead to a value higher than the actual basal rate (2) Radioactive iodine (I^{131}) uptake determination specifically measures the capacity of the gland to extract iodine from the circulating blood. A standard amount of I^{131} is ingested (about 50 μc) orally Twenty four hours later the neck is scanned, and the amount of radioactive material taken up by the thyroid gland is recorded The normal range of activity varies from 20 to 55 per cent uptake of the administered I^{131} (3) Conversion ratio or PBI^{131} adds to the information de-

rived from the uptake studies A sample of blood is taken 24 hr after a tracer dose Organic iodine is precipitated out and the specimen placed in a scintillation counter measuring the amount of ingested I^{131} that has been converted into thyroxin and discharged into the blood (4) Chemical PBI is probably the most direct measurement of circulating hormone However, the quantities are so minute that accurate determinations can only be done in ideally established laboratories by thoroughly trained personnel In general, hypermetabolic states tend to be associated with a low range of serum total cholesterol whereas hypometabolic states are usually associated with a hypercholesteremia These changes are not sufficiently sensitive to be of great help in evaluating thyroid function

HYPOTHYROIDISM

The function of the thyroid gland in maintaining a normal level of metabolic activity is dependent upon an adequate intake of the essential ingredient *iodine* In vast areas of the earth's surface the iodine content of the soil and drinking water is insufficient for this purpose and hypothyroidism is endemic. *This problem is intimately linked to that of* simple colloid goiter which is discussed later Other causes of hypothyroidism are pituitary hypofunction, thyroid aplasia, and inflammatory or surgical destruction of the thyroid substance

Hypothyroidism may exist as either *(1)* cretinism a result of congenital thyroid insufficiency or (2) adult, or acquired myxedema.

There is little difficulty in the diagnosis of cretinism with the characteristic picture *of the dwarfed bloated thick-skinned,* and mentally retarded infant The central nervous system damage is not reversible with thyroxin

Adult myxedema is consequent upon partial or complete suppression or destruction of the thyroid gland which may occur spontaneously or as a sequel to operative therapy It is characterized by a diminished basal *metabolic rate and by* a clinical picture showing edema of all body tissues. The stupid facies is a result of edema of the skin *and subcutaneous tissues, with loss of function* of the platysma, the muscle of expression Vocal cord edema results in a coarse thick voice *Laboratory findings in myxedema* are essentially a low BMR and little or no I^{131} uptake, with excretion of almost all administered *tracer doses within 3 days.* The conversion ratio of administered I^{131} similarly approaches zero Total circulating blood cholesterol is usually elevated. Anemia is characteristic, and the fires of life in general burn low Therapy of myxedema is not difficult, since most of the symptomatology is reversible with an adequate continuous supply of active thyroid principle

THE FOUR GOITERS

The term *goiter* is applied to a large variety of thyroid enlargements all of which are associated with metabolic disturbances. The thyroid enlargements of inflammatory or malignant disease are generally not included amongst the goiters Despite its multitudinous forms, all goitrous changes are *part of a simple physiologic cycle* that of repetitive hyperplasia and involution

Nontoxic Diffuse

Nontoxic diffuse goiter a deficiency state is a vanishing disease Formerly it was endemic *in distribution, in areas* where the drinking water contained a deficient amount of iodine Under such conditions the acini were distended with a homogeneous colloid material surrounded by a very low cuboidal epithelium The follicles and, subsequently the entire thyroid gland became markedly enlarged. The only real disability is pressure symptoms on vital structures within the neck and an undesirable cosmetic appearance Because of the histologic picture of the marked distention of the acini with colloid an alternate name for nontoxic diffuse goiter is *colloid goiter* There are no constitutional

signs and symptoms The incidence is increased at the time of puberty and is far more common in females than in males

Nontoxic Nodular

The term *nontoxic nodular goiter* is applied when there are one or more nodules palpable in the thyroid of a patient who is euthyroid or hypothyroid There is not perfect agreement on the origin of most of these nodules Some believe them to originate as fetal rests and thus consider them as adenomas This term means benign epithelial neoplasm Since the weight of opinion is that these changes are not neoplastic, the use of the term may be incorrect It is much more probable that the nodules are the result of frequent cycles of hyperplasia and involution in response to fluctuating needs for thyroid secretion in the presence of relative deficiency Hence they are better designated as *nodules* or *involutional bodies* Such nodules may be solitary or multiple However true benign epithelial tumors of the thyroid gland do occur and the differentiation between them and the involutional bodies is tenuous and difficult

Thyroid nodules frequently undergo nonspecific degenerative changes These run the gamut of liquefaction necrosis, cyst formation, hemorrhage, and calcification

It is the general consensus that the single nodule has a higher incidence of malignancy than the gland in which there are multiple nodules This may be more apparent than real, because the greater incidence of carcinoma in the solitary nodule may be due to the fact that malignant tumors are usually unicentric in origin and the nodule has been carcinomatous since its inception Statistics from different clinics vary with respect to the percentage of carcinoma found in nontoxic nodular goiter However the morbidity and mortality rate of removing such nodules from a thyroid is distinctly lower than the lowest incidence of carcinoma found in any large series, which varies from 4 to 17 per cent. Because of the rising incidence of carcinoma in thyroid nodules as goiter becomes less frequent, surgical excision of nontoxic nodules particularly of the solitary ones has become the accepted procedure

Activity of a thyroid gland or more particularly of a nodule in the gland can be assessed roughly by scanning the neck with a machine sensitive to the radioactive emanations of I^{131} following its oral administration Thus a nodule may be "cold" meaning inactive or it may be "hot" meaning active This is some aid in arriving at a decision to remove or not to remove a given nodule Since malignant nodules rarely are hormonally active the finding of a "cold" nodule leaves one with the possibility that a thyroid cancer may be present, whereas the "hot" nodule practically eliminates that possibility See Figure 21 for representative scintigrams

Occasionally a nontoxic nodular goiter will grow inferiorly below its usual resting place on both sides of the trachea and in front of it Such growths usually are not of normal thyroid tissue but are hypertrophic or hyperplastic. The growth is along the trachea, usually in the anterior mediastinum, and constitutes the entity of *substernal goiter* The blood supply of a substernal goiter is usually the inferior thyroid artery although occasionally the thyroidea ima, arising from the great vessels in the mediastinum, may contribute a substantial portion of the blood supply This is not to be confused with the occasional *intrathoracic thyroid* which is found in various regions of the chest and has no connection with the normal cervical thyroid.

Toxic Nodular (Hyperthyroidism)

Toxic nodular goiter refers to a thyroid gland with nodules and the clinical picture of hyperthyroidism. Such a clinical picture may be due to either of two sets of circumstances (1) the occurrence of hyperthyroidism owing to overactive internodular tissue and the incidental presence of "cold" involutional nodules (2) the more rare occurrence of hyperfunctioning adult adenomas. Such adult functioning adenomas are

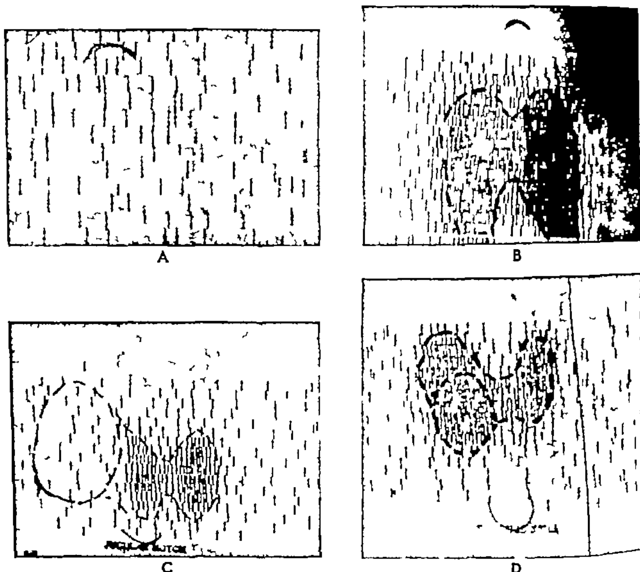


FIG 21 Thyroid scintigrams following administration of I^{131} *A* Very low uptake. *B* Diffuse high activity *C* A "cold" nodule *D* A "hot" nodule. (*Courtesy Department of Metabolism and Endocrinology The Michael Reese Hospital*)

also found in all other glands of internal secretion The indication for therapy in this disease is therapy of the hyperthyroidism Surgical excision is generally favored over isotope therapy in the toxic nodular goiter for two reasons First, the nodular gland is less responsive to isotope irradiation than is the diffuse goiter Second after the hyper functioning cells have been destroyed by I^{131} there is still the presence of a nodule in the thyroid gland which of itself often constitutes an indication for surgical excision Malignancy in a functioning adenoma although occasionally described is rare The detection of toxicity i e hyperthyroidism, by clinical and laboratory methods has been described

Toxic Diffuse (Hyperthyroidism)

Toxic diffuse goiter is a disease of unknown cause not originating within the thyroid gland but probably within the pituitary control over it or in higher centers. It is characterized by hyperfunction of the thyroid gland with remissions exacerbations, and crises by eye signs and by numerous systemic manifestations. The symptomatology characteristic of this disease largely results from the increased rate at which energy is transformed in the body resulting in an increased total metabolic rate Not all the characteristic symptomatology in the spontaneous hyperthyroid state can be reproduced by thyroid substance administration

Grossly the thyroid gland is usually symmetrical somewhat enlarged and firm The vascular supply to and blood flow through the gland are markedly increased Histologic examination of such a gland reveals both hypertrophy and hyperplasia of epithelial columnar cells in the extreme resulting in papillary formation There is only a small amount of poorly staining colloid There is a diffuse lymphocytic infiltration This histologic picture is reversible with the administration of iodine Under such conditions involution takes place the high columnar epithelium becomes flattened toward the cuboidal state there is an increase in the amount of colloid which stains more deeply the papillary folds are diminished or disappear the vascularity and lymphocytic infiltration in the gland are markedly reduced

For some unexplained reason the disease is not so common as it was 15 to 20 years ago Although it may occur at any age it is most common in the female in the third, fourth and fifth decades of life The only environmental change upon which this could be explained is the widespread use of iodine in salt or water

The patient commonly presents herself with the following symptoms (1) progressively increasing nervousness with inexplicable outbursts of crying she is short tempered and irascible with her husband and children without unusual provocation there may be purposeless movements and inability to sit still for more than a few moments there may be a subjective feeling of trembling All these symptoms may come under the heading of nervous instability There is further (2) an increased appetite (3) weight loss despite the increased food intake (4) an increased amount of perspiration, with an intolerance of heat, (5) shortness of breath, (6) palpitation, (7) muscular weakness (8) diarrhea Examination of the patient reveals the following outstanding findings (1) the patient s nervousness is obvious to the examiner one usually sees a demonstration of crying without provocation and also rapid, jerky purposeless movements of the extremities and, similarly a staccato type of speech (2) tachycardia tachypnea elevated blood pressure and pulse pressure (3) a fine tremor of the hands (4) a rather firm gland usually enlarged (5) frequently an exophthalmos (6) a bruit or thrill over the neck (due to the tremendous increase in vascularity and blood flow of the thyroid gland) (7) frequently a cardiac irregularity which may be auricular fibrillation (8) cardiomegaly (9) cardiac decompensation in the extreme case (10) laboratory findings of (*a*) increased basal metabolic rate (*b*) increased cardiac output (*c*) lowered serum cholesterol concentration (*d*) elevated radioactive iodine uptake and (*e*) an elevated PBI and conversion ratio

In the differential diagnosis of hyperthyroidism must be included psychic factors which may produce all or any part of the above symptomatology and all other diseases which produce tachycardia and muscular weakness and an elevated BMR and I[131] uptake The therapeutic test with the administration of iodine becomes a differentially diagnostic test, in that the hyperthyroid patient will usually respond by improvement and other causes simulating the disease will not

The therapy of toxic diffuse goiter has been in a state of flux because of the addition of new modalities of management. In general there are three approaches medicinal isotopic, and operative

Drug therapy may be definitive or adjunctive to surgical or even to isotopic management. The *antithyroid drugs* of the thiourea group prevent the synthesis of thyroxin within the gland By so doing, they are able to bring the hyperthyroid patient to a euthyroid state In young patients and in borderline thyrotoxicoses such remission may be permanent, with or without a long-continued maintenance dose In most instances, however the remission is temporary and is utilized as a preoperative preparatory measure These drugs are responsible for the production of a severe anatomic hyperplasia which

may add difficulty and danger to subsequent thyroidectomy If they are employed as a preoperative measure involution of the gland must be accomplished with the administration of iodine for at least 10 days prior to anticipated surgical therapy The antithyroid drugs are toxic to a greater or less degree, and their administration may be attended by an appreciable incidence of agranulocytosis They must always be administered with caution and with frequent determinations of the white blood cell count Additionally there may be a recurrence of the hyperthyroid state immediately after discontinuance of these substances there may even be an escape from their suppressing action while they are being administered

Radioactive iodine is being used increasingly frequently in the therapy of hyperthyroidism. It is much more effective than external irradiation which was often used 15 or 20 years ago particularly in patients who could not be properly prepared for surgical therapy I^{131} has a half life of 8 days Its beta radiation is effective only for a distance of 2 or 3 mm but its gamma emission can be measured external to the body this is basically the way in which its effectiveness is determined by scintillation counters The beta radiation of I^{131} is responsible for the amelioration of the hyperthyroid state This is indeed pinpoint radiation, since the malignant cell picks up its own lethal molecule of I^{131} and commits suicide A varying amount of fibrosis in the gland automatically follows its use If given in sufficiently large quantities total destruction of the gland may occur with the production of myxedema. This is no great catastrophe since myxedema is much more readily treated in the adult than is the original hyperthyroidism It is well known that under certain conditions beta radiation may be carcinogenic Whether I^{131} has ever actually produced a carcinoma of the thyroid gland, and how long it takes for this transformation is not really known at the present time In all probability such manifestations may not occur in less than 20 or 25 years Largely for this reason the therapy of hyperthyroidism with this radioactive compound is usually reserved (1) for those at the age of forty or older and (2) for conditions where the patient cannot be properly prepared for surgical therapy because of some other pre-existing disease I^{131} must not be used in the pregnant female because of its effect on the thyroid of the fetus It has recently been shown experimentally that the administration of I^{131} resulted in cretinous offspring in the laboratory animal

An alternative method for the treatment of hyperthyroidism with a diffusely involved gland is *surgical extirpation* of most of the thyroid gland This is the method preferred by most surgeons Inorganic iodine and the antithyroid drugs are useful and frequently necessary in adequate preoperative preparation of the patient. It is the consensus of surgeons that radioactive iodine should be used by choice only after the age of forty

The preoperative preparation of the hyperthyroid patient is all important. (1) The BMR must be brought not necessarily to the lowest possible level but to a stable lowered level In the mild hyperthyroid patient this may be done with inorganic iodine (Lugol's solution) saturation and maximal response is usually reached in 20 days The administration of inorganic iodine causes an involution of the extreme hyperplasia of the thyroid gland causes the specific hormone to be stored in the colloid of the acini and prevents the further action of this substance When the patient presents herself with markedly elevated basal metabolic rate (anything over BMR of 50) the antithyroid drugs (thiourea) may be very useful Propylthiouracil will usually result in a reduction of the BMR from such levels by about 0.5 per cent per day Frequent determinations of the white blood cell count including a differential count must be made Escape from the effect of the drugs, even during administration must be occasionally expected (2) A high caloric diet must be provided preferably with a high protein intake An extremely important criterion of the ef

fectiveness of increased caloric intake is weight gain An adequate fluid intake must be a part of the preoperative preparation, in order to provide sufficient urinary output to dispose of an increased production of tissue metabolites. (3) Adequate rest is mandatory particularly for (4) the therapy of the associated cardiac disease Meticulous attention must be paid to the therapy of cardiac decompensation with or without auricular fibrillation The patient's ultimate prognosis may depend on how much cardiac damage is done and to what degree it is reversible before surgical therapy (5) There must be adequate therapy of a commonly associated diabetes mellitus, hyperthyroidism frequently being one facet of a multiglandular endocrinopathy (6) Vigorous therapy of thyroid crises which are not as commonly seen as in bygone years must be provided. Such therapy includes (*a*) the intravenous administration of sodium iodide in 2-Gm doses, (*b*) ice packs to combat the profound hyperthermia, (*c*) sedation to combat the extreme central nervous system irritability (*d*) mercurial diuretics and (*e*) full digitalization for preservation of maximum cardiac function The corticosteroids, which have a marked antiinflammatory action, may be very helpful

The operative goal in the therapy of the hyperthyroid state is the reduction of the amount of thyroid substance in the body to the point where excessive thyroxin output is no longer possible This should be accomplished under conditions that are optimal, both physically and mentally for the patient The prognosis of this form of therapy depends on (1) the amount of tissue removed, (2) the degree of damage already inflicted on vital organs, and (3) the persistence of the pituitary or other factors that originated the disease in the first place

There are three potential complications in the *early* postoperative period. Hemorrhage with resultant tracheal compression, must be guarded against with meticulous hemostatic technic. Only a small bandage should be applied to the wound postoperatively so that all attendants may observe any evidence of gross bleeding should it occur *Respiratory embarrassment* must be detected as soon as it occurs. Respiratory stridor may also signify the compromising of one or both *recurrent laryngeal nerves* The occurrence of bilateral paralysis, due either to cutting of the nerves at the time of operation or to a temporary edema, with the approximation of the cords (cadaveric position) may be a genuine emergency Under such conditions a tracheostomy must be performed without delay For facility a tracheostomy set should always be available or the night table at the patient's bedside The incidence of injury to the recurrent laryngeal nerves is greater during secondary explorations of the neck than upon initial surgical procedures *Parathyroid tetany* due to acute hypoparathyroidism, consequent upon removal of all four of the parathyroid glands in the neck (or their temporary physiologic suppression by edema or hemorrhage) must be anticipated if the thyroid resection is radical or if the glands are found upon subsequent examination by the pathologist The symptoms and signs of profound muscular irritability must be detected early Therapy is adequate with the administration of dihydrotachysterol calcium, and vitamin D Usually when edema subsides or when other parathyroid tissue (for example, in the mediastinum) assumes its function, the symptoms of hypoparathyroidism will abate

Late complications following surgical therapy of hyperthyroidism are as follows (1) There may be recurrence of the hyperthyroidism which may largely be prevented if inorganic iodine is used postoperatively for a few weeks (2) Occasionally a preexisting exophthalmos may be aggravated after the removal of a large thyroid gland. This is probably due to the removal of the inhibitory influence of thyroxin on the anterior pituitary substance which is responsible for exophthalmos. The exophthalmos-producing hormone of the anterior pituitary is secreted parallel with TSH, but can be demonstrated as a separate substance In

creased output in exophthalmic goiter explains exophthalmos as a symptom The further stimulus to the anterior pituitary of the hypothyroidism following therapy explains the progressive exophthalmos seen postoperatively Some surgeons believe the exophthalmos itself locally to be due to an increased amount of edema fluid in the orbit others attribute it to a hypertrophy of the muscles which produce proptosis At all events the administration of desiccated thyroid substance in the postoperative period will prevent the recurrence of or at least the exacerbation of the proptosis (3) Myxedema which is easily treated with thyroid substance may occur

THYROIDITIS

Thyroiditis is an inflammatory disease of the thyroid gland which occurs in a number of forms It may be acute subacute, or chronic, and three rather distinct types of the subacute-chronic variety are seen clinically and described pathologically

Acute

Acute thyroiditis may occur as an isolated finding often it is associated with infection elsewhere in the body It is characterized by local swelling tenderness and sometimes hyperemia of the overlying skin. Systemic manifestations may be severe and include chills and fever The disease is usually of short duration and subsides spontaneously However occasionally suppuration occurs, necessitating drainage

Subacute and Chronic

The most common of the subacute-chronic type of thyroiditis is the nonspecific, or granulomatous form it is a disease the cause of which is not entirely clear but it is frequently seen following or as a part of systemic virus disease The pathologic findings are enlargement of the thyroid, which is usually smooth although it may contain multiple small nodules Histologically there is evidence of hypertrophy and hyperplasia a marked increase in vascularity a spectacular increase in the number of leukocytes in the gland especially lymphocytes foreign body giant cells and often granulomatous bodies Because of the presence of giant cells this disease is sometimes called *giant-cell* or *pseudotuberculous thyroiditis* Clinically the presenting symptoms are soreness in the neck, pain extending to the ears and jaws being occasionally accompanied with difficulty in swallowing or in breathing and fever Examination of the neck reveals the thyroid to be enlarged frequently harder than usual and very tender to palpation The nodular structure is sometimes discernible In addition to these local signs of inflammation there are the systemic findings of fever and leukocytosis The therapy of the disease is similarly nonspecific corticosteroids are helpful in reducing the inflammatory process X radiation and the antithyroid drugs are occasionally helpful as is TSH. Hypothyroidism may follow any inflammatory disease of the thyroid if the acinous tissue is destroyed

Hashimoto s struma or *struma lymphomatosa* has been authentically described in females exclusively The symptomatology and findings are not unlike thyroiditis of other origin The gland may be described pathologically as being inundated with lymphocytes so much so that it appears almost as if it were a lymph node The involvement may be patchy or diffuse. Recent investigations have revealed an autoimmunization of the serum in cases of Hashimoto s disease to the thyroid tissue. There is characteristically an increase in the gamma globulins, often sufficient to reverse the albumin/globulin ratio The gamma fraction of the globulins is known to carry immune bodies Operative therapy is employed for diagnosis only and for relief of tracheal symptoms. The thyroid should not be excised since the disease itself tends to leave a hypothyroid state in its wake.

Riedel's struma is another form more specific pathologically but also of unknown cause It is also referred to as *ligneous thy*

roiditis because of the stony, hard character of the gland The clinical symptoms and signs are similar to those of carcinoma except that there are more symptoms of trachea The reason for this is not difficult to see on histologic examination of the gland which gives the appearance of strangulating thyroid tissue It would appear that strangulation of thyroid cells actually occurs since a mild hypothyroid state is the rule Operation is indicated only to make a diagnosis and to relieve pressure on the trachea. This is accomplished with a small biopsy and occasionally by cutting or removing the isthmus One must be careful when this pathologic diagnosis is made not to remove too much glandular tissue lest myxedema ensue Adjunctive supportive measures are indicated.

CARCINOMA

Incidence and Etiology

The incidence of malignant tumors of the thyroid gland found in surgical material varies with many factors The age incidence in general is that of carcinoma anywhere although carcinomas of the thyroid in childhood are not uncommon particularly in females The incidence will further vary with the nature of the practice of the reporting group, being higher in referred practices than in a true cross section of the population or of hospital practice

Almost all malignant tumors of the thyroid gland are epithelial in nature (carcinomas) with only an occasional connective tissue malignancy (sarcoma) Most neoplasms arise from adult epithelium however it is the opinion of some pathologists that some arise from fetal epithelium, i e., from a true fetal adenoma.

Pathology

Pathologically the following types are described (1) differentiated carcinomas, which may be (*a*) papillary adenocarcinoma or (*b*) follicular carcinoma (2) undifferentiated carcinomas The differentiated carcinomas are in general of low grade malignancy although the lymph channels to the adjacent and subjacent areas of the neck may be involved as the only evidence of spread hematogenous metastasis may occur this finding is usually a late one in terms of years Metastases to bone lung, and liver are most common with the undifferentiated variety Metastases may be found anywhere from 10 to 25 years after the initial discovery of a papillary adenocarcinoma of the thyroid The undifferentiated carcinomas are much more rapidly fatal Hürthle cell carcinoma, a pathologic entity questioned by some pathologists is seemingly also of low-grade malignancy

Diagnosis

The diagnosis of carcinoma of the thyroid is usually difficult. It must be suspected in any nodule of the thyroid particularly if solitary As goitrous nodules become progressively less frequent, the relative incidence of carcinoma in thyroid nodules increases. At the present time the incidence is sufficiently high that it has become generally accepted that all such masses, particularly if nontoxic and especially if single should be removed and sectioned. Possible exception to this rule is the presence of a nodule in a patient over the age of fifty In whom the nodule has not changed for many years even this exception is questioned by some Two screening devices, beyond the history and physical character of the mass are being used Radioiodine uptake within the nodule as demonstrated by the scintogram (scanning of the cervical region to map out activity of various portions of the thyroid tissue) is the first method The so-called "warm," or "hot," nodule speaks strongly against carcinoma, because most malignant tumors are physiologically inactive A second method is the response to thyroid extract or hormone used as a therapeutic test If the mass shrinks notably in size it is most unlikely that it is cancer Compression of the trachea, like remote

metastases carries a high index of suspicion, but then it is too late for effective therapy

The presence of any discrete lymph node in the neck whether near or far from the thyroid gland also requires examination by excision biopsy The finding of what has in the past been called *lateral aberrant thyroid* i e papillary thyroid tissue within the lymph node even if histologically benign is now taken, by concensus to indicate metastasis from a papillary carcinoma in the ipsilateral hemithyroid Careful histologic examination of that thyroid lobe will not fail to reveal the primary tumor

Treatment

The management of the patient with carcinoma of the thyroid gland is in a highly controversial state at the present time It is generally agreed that surgical extirpation is the most effective measure, but the indications and extensiveness of the operation are moot The problem is aggravated by the extremely wide range of malignancy corresponding in a very general way with the prevailing cellular type of tumor The *papillary adenocarcinomas* which constitute more than half of all thyroid cancers are relatively benign and slow-growing They metastasize late and, characteristically to the regional lymph nodes. Even after metastasis has occurred long survival and sometimes permanent freedom from recurrence may be expected The lateral aberrant type, mentioned above falls into this category It is believed by most authorities that hemithyroidectomy and radical dissection of the neck on the involved side should be performed A similar relatively benign type is the Hürthle cell carcinoma. This is rare and the management is similar to that of the papillary variety

Follicular carcinoma is far more malignant and it early becomes disseminated by way of the blood stream Local excision and lymph node dissection are therefore much less effective Nevertheless total excision of the involved lobe and isthmus plus subtotal removal of the opposite lobe and unilateral or bilateral radical neck dissection is the program usually followed *Undifferentiated carcinoma* is extremely malignant and is almost invariably fatal in 6 to 12 months, irrespective of the therapy employed.

Adjuvant treatment particularly with therapeutic doses of I^{131} is of some value But its usefulness is limited to those tumors primary or metastatic that are sufficiently differentiated to be physiologically active enough to take up the lethal molecule of radioactive iodine The over all incidence of such activity is about 15 per cent of all thyroid carcinomas To a degree however uptake can be stimulated by measures which promote cellular hyperplasia. These include removal or destruction of all normal thyroid tissue to render the patient hypothyroid and then an increase in the output of autogenous TSH of the anterior pituitary gland will take place This hormone may be given pharmacologically as may also antithyroid drugs. Metastatic lesions, which are originally functionally inactive may sometimes be made to take up enough I^{131} to destroy themselves This is particularly true in the follicular type External irradiation has not proved of sufficient value to merit its use beyond palliation

MISCELLANEOUS LESIONS

The migration of the embryonic thyroid elements from their anlage makes possible the arrest of descent and the appearance of thyroid tissue in other than the usual adult position Since the midline bud of the thyroid embryologically came from the endoderm and descended from the position of the foramen cecum at the base of the tongue down toward its final resting place, some thyroid tissue may be left at the base of the tongue which may be discovered in adult life This *lingual goiter* is usually removed with ease Reference is made to Chapter 23

In the descent of the thyroid from the foramen cecum to its adult position in the

neck a sinus tract may be left patent which may terminate in the midline of the neck just inferior to the hyoid bone The patency of this sinus tract in its communication with the oropharynx permits infection A midline swelling in the neck, usually just inferior to the hyoid bone, is an infection of this sinus tract It is known as a *thyroglossal duct cyst sinus* and usually traverses the hyoid bone Therapy is by excision of the entire sinus tract from the skin of the neck to and including the foramen cecum at the base of the tongue Reference is made to Chapter 14

SUMMARY OF PRINCIPLES

1 The thyroid is a gland of internal secretion richly endowed with blood supply and intimately related to other important structures in the neck.

2 The hormone of the thyroid gland thyroxin is essential for the metabolism of all body cells To manufacture this product the thyroid gland selectively removes iodine from the circulating blood The thyroid gland is not self-contained and autonomous it is part of a complex interglandular relationship involving other endocrines particularly the anterior pituitary

3 Hypothyroidism may be congenital or acquired its treatment is not difficult although the central nervous system damage in cretinism is not reversible

4 Nontoxic diffuse, or simple colloid goiter is a vanishing disease. It is due to an insufficient supply of iodine in the soil and in drinking water

5 Nontoxic nodular goiter is the presence of nodules in the thyroid gland in the presence of a euthyroid or hypothyroid state It may be due to repeated cycles of hyperplasia and involution with secondary changes such as cyst formation hemorrhage and calcification.

6 Toxic nodular goiter is the presence of hyperthyroidism in a patient whose thyroid gland contains one or more nodules The hyperfunctioning thyroid tissue may or may not be within the nodules

7 Toxic diffuse goiter (Basedow's disease Graves s disease hyper thyroidism with or without exophthalmos) is a disease which does not originate within the thyroid gland but probably within higher centers It is characterized by systemic symptoms and signs which are related to the increased rate at which energy is transformed in the body The usual therapy includes accurate preoperative management of the patient surgical excision of the thyroid gland, the avoidance of complications, and the therapy of tissues already damaged by the high metabolic rate An alternate method of therapy in the age group over forty is the administration of radioactive iodine (I^{131})

8 The preoperative use of the antithyroid drugs prevents the formation of thyroxin. The use of inorganic iodine aids in involution of extreme hyperplasia

9 Postoperative thyroidectomy complications include (*a*) hemorrhage (*b*) tracheal compression (*c*) respiratory embarrassment due to recurrent laryngeal nerve damage or edema, (*d*) hypoparathyroid tetany

10 Exophthalmos is probably due to a substance formed in the anterior

pituitary which is inhibited by normal thyroid secretion. Postoperative exacerbation of exophthalmos is prevented by the postoperative use of desiccated thyroid substance and inorganic iodine

11 Thyroiditis is an inflammatory disease of the thyroid gland which may be acute subacute, or chronic There are three main varieties of the subacute-chronic type Nonspecific or giant-cell thyroiditis is of unknown origin possibly viral Riedel s struma, or ligneous thyroiditis presents a characteristic histologic picture and may lead to tracheal compression Hashimoto s struma or struma lymphomatosa is described exclusively in females and may be due to autoimmunization.

12 Malignant tumors of the thyroid gland are almost all epithelial in nature

13 Lateral aberrant thyroid tissue is really a metastatic lesion to a primary tumor of the thyroid lobe on that side

14 Carcinoma of the thyroid gland is treated by surgical excision or, occasionally by the use of radioactive iodine.

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14

Masses and Swellings in the Neck

I **Masses and sinuses of embryonal remnants**
  A Branchial pouches and clefts cysts, sinuses, and tumors
  B Thyroglossal duct cyst and sinus
  C Angiomas
II **Thyroid**
III **Lymph nodes**
  A Lymphoma group (primary malignant disease) (1) lymphosarcoma (2) Hodgkin's disease
  B Metastatic malignant disease (1) from head and neck (2) from thoracic and abdominal viscera
  C Inflammatory disease (1) acute (*a*) adenitis (*b*) abscesses (2) chronic (*a*) tuberculosis (*b*) sarcoidosis
IV **Abscesses and cellulitis**
  A Origin in floor of mouth
  B Origin in mediastinum
V **Salivary glands**
  A Tumors
  B Infections
VI **Vascular and paravascular enlargements**
  A Aneurysms
  B Carotid body tumors
VII **Tumors of the mandible**

MASSES AND SINUSES OF EMBRYONAL REMNANTS

Branchial Pouches and Clefts; Cysts, Sinuses, and Tumors

Early in embryologic life there develops in the cephalocervical region five sets of arches and clefts from which develop various structures of the head neck, and upper mediastinum The arches become cartilaginous and give rise to skeletal structures the clefts, or furrows occupy the spaces between the arches and present external and internal surfaces From the first arch come the upper and lower jaws and two of the bones of the middle ear The second arch gives rise to the styloid process, the hyoid bone, and the third bone of the middle ear From the third arch is derived a part of the hyoid bone The fourth furnishes the *anlage* for most of the thyroid cartilage and the fifth provides the remainder of the thyroid cartilage as well as the arytenoid and cricoid cartilages The first branchial furrow (cleft) forms a tubular structure *on its lateral side* which usually becomes obliterated except for its dorsal portion which persists as the *external* auditory meatus The visceral side of the cleft, or the visceral furrow develops into the eustachian tube and the *tympanic*

cavity The second branchial furrow usually is completely obliterated. The third branchial furrow gives rise to the thymus and the inferior parathyroids The fourth branchial furrow develops into the superior para thyroids. Anomalies in this embryologic process lead to a number of abnormal con ditions which may not become manifest until later in life

The lateral tube of the first branchial furrow occasionally remains patent in its prox imal portion This leaves an epithelium-lined tract which has no external outlet. Distention of this tract with fluid secretions converts it into a cyst. If it opens onto either a mucous or cutaneous surface a *sinus* exists if it opens onto both surfaces, a *branchial fistula* is present Similar failure of obliteration of the second furrow occurs much more frequently than of the first and also results in cysts, sinuses, or fistulas Branchial cysts from either of these furrows generally present at the angle of the mandi ble those from the first extend toward the external auditory meatus, and if a sinus de velops, it opens into the auditory canal. Cysts from the second furrow are traceable toward the tonsilar fossa, and sinuses may open at this point External openings of these sinuses (or fistulas, if open at both ends) are found at the angle of the jaw or in the lateral surface of the neck as far down as the clavicle These *branchial cysts* and *sinuses* are almost always benign Oc casionally a malignant tumor grows from the epithelium These are known as *bran chiogenic carcinomas*

Thyroglossal Duct Cyst and Sinus

The thyroid gland develops from an evagination in the floor of the pharynx, a site which later becomes the foramen cecum of the tongue This anlage travels caudad usually through the hyoid bone and comes to rest on the anterior surface of the trachea whence it extends to both sides to form the thyroid lobes The tract that is left behind the descent of the thyroid gland is normally obliterated When it remains open in its entirety or in part, a sinus tract lined by epithelium persists As in the case of the branchial derivatives either the internal or the external end, or both may be open. Such sinus tracts (*thyroglossal duct sinus*) may lie anywhere in the midline between the foramen cecum and the skin of the anterior surface of the neck as far down ward as the isthmus of the thyroid gland, and they usually pass through the hyoid bone (Fig 22) *Thyroglossal duct cysts* are

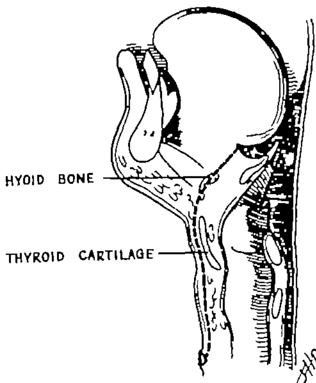


FIG. 22. Thyroglossal duct sinus and/or fistula. The curved line traces the pathway from the foramen cecum at the base of the tongue through the hyoid bone in front of the thyroid cartilage and erupting onto the skin in the midline of the neck.

circumscribed swellings in the midline of the neck resulting from portions of this tract which are closed at both ends. Any cyst or sinus opening in the midline of the neck is probably of thyroglossal duct origin they should not be mistaken for simple epidermoid cysts The only complete and effective therapy of such cysts and sinuses is complete excision of the entire tract in cluding the central portion of the hyoid bone up to and including the foramen cecum

of the tongue Less than this invites recurrence, usually in the form of an abscess and dissecting sinus tract

Angiomas

Angiomas of the neck may be either hemangiomas or lymphangiomas. Of the *hemangiomas* both the capillary and the cavernous types are seen These vascular nevi may usually be removed by x radiation, radium therapy cryotherapy (cold therapy carbon dioxide snow) or wide excision sclerosing agents are still used for the obliteration of some of these lesions. *Lymphangiomas* are also of the capillary the cavernous and the cystic types Almost all are found in early childhood The most spectacular the *cystic lymphangiomas* or *cystic hygromas* are usually found at the lateral border of the sternocleidomastoid muscle and may be very extensive reaching from the skull to the clavicle Compression of cervical viscera may result Therapy consists of wide surgical excision of this multilocular lesion, usually preceded by some type of irradiation to shrink the tumor The excision of the larger cystic hygromas entails considerable morbidity because of their intimate relation to the major vessels and nerves in the neck. Recurrence is due to failure to remove the complete lining of the cyst.

THYROID

Tumors nodules, and swellings of the thyroid gland loom prominently in the differential diagnosis of masses in the neck. Reference is made to Chapter 13 Particular mention is made of so-called *lateral aberrant thyroid* When a small tumor is found in the lateral reaches of the neck, that is, outside the usual confines of the thyroid gland, which upon biopsy is reported histologically to be thyroid tissue it is commonly referred to as lateral aberrant thyroid tissue Such rests were formerly thought to be normal thyroid tissue in an aberrant position but it is now generally agreed that they are really metastatic carcinomas in regional lymph nodes from the ipsilateral lobe of the thyroid gland

LYMPH NODES

Lymphoma Group (Primary Malignant Disease)

Malignant tumors of lymphoid structures constitute connective tissue neoplasms or sarcomas *Lymphosarcoma* may be found in lymphoid tissue anywhere in the body It is usually *multicentric* that is occurring in many places in the body simultaneously although it may be detected in but one group of lymph nodes The various chains of lymph nodes in the neck constitute a common site for the appearance of this lesion Lymphosarcoma occurring in many places in the body is called *lymphosarcomatosis* The demonstration of lymphosarcoma in the blood stream has been designated *leukosarcoma* Histologically three types are differentiated (1) lymphoblastoma, (2) lyphocytoma, and (3) reticulum cell sarcoma.

The diagnosis of lymphosarcoma is usually made on the basis of biopsy of lymph nodes which are accessible and palpable or roentgenologically demonstrable (mediastinum) the occurrence of otherwise undiagnosed fever and other symptoms depending on the location of the masses Local excision of these masses is only rarely of value and treatment is systemic, including irradiation tumor-destroying chemicals and radioactive materials

Other malignant tumors of lymphoid tissue, *malignant lymphomas* are usually considered to be neoplasms, although their exact origin is not known. They present characteristics of true neoplasms in some respects but there remains the possibility that viruses may be the causative agents producing their growth

Hodgkin's disease is a generalized condition in which lymphatic structures throughout the body are enlarged and pre

sent a characteristic histopathologic picture, it leads usually to a fatal outcome It may be found at any age and in either sex although it is more frequent in early adulthood It is commonly detected first in the cervical lymph nodes. The pathology varies with the stage of the disease Early only *lymphoid hyperplasia* is seen which may be so extensive that the normal architecture of the lymph node is lost At a later stage the characteristic Sternberg Reed cells a modified type of foreign body giant cell, associated with eosinophils are characteristically seen Early the lymph nodes are rather discrete later with fibrosis, the glands become matted together Four stages or types of the disease are described The earliest stage often takes the form of *giant follicular hyperplasia* (Brill-Symmers disease) This condition usually eventuates in one of the several forms of malignant lymphoma. The next pathologic stage described resembles a granuloma more than a neoplasm and has been called *Hodgkin s granuloma* when the nodes exhibit granulomatous changes but also some that are characteristic of neoplasia, the term *Hodgkin s paragranuloma* is applied When clearly neoplastic changes are obvious especially when invasion of adjacent tissues takes place the term *Hodgkin s sarcoma* is appropriate

The diagnosis of Hodgkin s disease is made on the history of an insidious onset of malaise and weakness fever of the Pel Ebstein type and the biopsy of palpable lymph nodes usually in the neck and frequently in the mediastinum These nodes are usually painless The blood picture usually includes leukopenia (usually a lymphopenia) moderate eosinophilia a relative monocytosis and anemia. The spleen is palpable in about 50 per cent of cases

Treatment of Hodgkin s disease is most unsatisfactory and a relentless downhill course is characteristic although the duration may extend over a period of 10 or more years Local excision is not curative irradiation of involved areas is sometimes helpful and the use of chemical agents such as the nitrogen mustard group is also sometimes of benefit.

Lymphatic leukemia is a blood dyscrasia in which there is a marked increase in the number of circulating lymphocytes in the blood stream and in which there is also a cervical lymphadenopathy The nodes usually do not become adherent to each other and they remain soft. Histologic examination reveals lymph nodes that are replaced by lymphocytes

The acute lymphatic leukemias are seen in young children and the chronic lymphatic leukemias are seen at the other extremity of life in the aged The accompanying anemia in leukemia is due to an encroachment on the erythropoietic system (myelophthisis) Acute lymphatic leukemia is not successfully treated the duration of life in chronic lymphatic leukemia may be very long Irradiation and palliation with nitrogen mustard and radioactive phosphorus are frequently helpful

Metastatic Malignant Disease

The diagnosis of malignant disease remote from the neck is not uncommonly made by biopsy of a painless lymph node in the cervical region. Metastatic involvement of both deep and superficial chains of nodes is not uncommon in malignancies of the mouth nasopharynx, oropharynx and thyroid and even of the larynx before the areas in which the tumors originated become symptomatic Before excision of such a node for biopsy all of the above-mentioned areas must be carefully surveyed for primary growth.

Metastatic malignant involvement of the lymph nodes of the neck is also very common in bronchogenic carcinoma. The nodes that are usually involved earliest are those of the scalene group in the supraclavicular area Biopsy of a scalene node when bronchogenic carcinoma is suspected is of more diagnostic value in general then bronchoscopic examination in the same patient The finding of bronchogenic carcinoma in a scalene node does not necessarily mean the

presence of cancer in the lung on the ipsilateral side because the lower lobe of the lung is frequently drained by lymph channels which cross over to the right paratracheal and then up to the right scalene area. Gastric neoplasm is occasionally detected by a left supraclavicular lymph node (*Virchow's node*) The cervical nodes may be involved in cancer of the esophagus although usually this disease becomes manifest by direct extension before it becomes metastatic

Radical neck dissection is the term used for the wide removal of the lymph nodes of the neck in order to extirpate cancer metastases, whether gross or microscopic. This operation is attempted usually only for those malignant tumors which originate in the head and neck and not for those which are of remote origin. Thus, one would attempt a radical neck dissection for a carcinoma of the floor of the mouth or of the thyroid gland but not for metastatic carcinoma from the lung or stomach Such node dissections require the removal of the deep system of lymph node chains as well as the superficial This necessitates not only the excision of the sternocleidomastoid muscle with its superficial chains and its enveloping fascia, but also the removal of the deep chain in and around the carotid sheath and frequently a suprahyoid dissection for the removal of salivary glands and lymph nodes in the submaxillary region The internal jugular vein must be removed from the clavicle to its point of emergence from the cranium This entire dissection is done preferably en bloc At the completion of such a dissection there should remain in the neck, carefully preserved, the following structures the vagus nerve the sympathetics immediately posterior to the carotid sheath, the carotid artery the prevertebral muscles the phrenic nerve, and the spinal accessory nerve When bilateral radical neck dissection is deemed advisable an interval is usually allowed to elapse between performance of this operation on the two sides since bilateral removal of the internal jugular veins in man in one operation is not well tolerated because of cerebral congestion and edema as a result of inadequate venous drainage

Inflammatory Disease

Inflammatory or infectious disease of lymph nodes, *lymphadenitis* may be acute or chronic

Acute pyogenic adenitis is seen in the neck as anywhere else in the body as a first response to local infections These usually originate in the adjacent skin ears scalp or oropharynx. Examination of the head and neck for primary infections will usually uncover the source Surgical treatment of the local infection incision and drainage of suppurating nodes, and the use of systemic antibiotics usually result in a successful short clinical course

Chronic cervical lymphadenitis may be nonspecific or tuberculous Tuberculosis of cervical lymph nodes, formerly very common and now relatively rare, must still be dealt with occasionally here and very frequently in many parts of the world It is most common in the early years of life and frequently is associated with contact with tuberculous patients or the ingestion of unpasteurized milk *Tuberculous adenitis* in the young may rather rapidly become caseous and then purulent, with the usual signs of fluctuation and local heat. Thorough study of the entire patient for tuberculosis, as well as culture of the material from the adenitis must not be omitted Chronic nonsuppurative adenitis, especially in the earlier years of life may require excisional biopsy for diagnosis All systemic therapeutic measures effective in tuberculosis should be employed. The role of surgery in tuberculous cervical adenitis is restricted to (1) biopsy for diagnosis (2) drainage of abscess and (3) excision of sinuses.

Occasionally difficult of differential diagnosis from tuberculosis in the neck is *sarcoidosis* (*Boeck's*) In sarcoidosis there may be diffuse adenopathy most commonly in the mediastinum and neck, but there may also be involvement of many other viscera,

particularly the spleen and the reticulo-endothelial system of the liver There may even be lesions of the small bones of the hands and feet, which if present, aid in *diagnosis* *The cause of sarcoidosis is not agreed upon* some investigators believe it to be a modified form of tuberculosis others *believe it is more closely related to the* hyperimmune group of syndromes

Syphilis may occasionally lead to a generalized adenopathy The diagnosis can be made by biopsy in conjunction with other systemic diagnostic procedures

ABSCESSES AND CELLULITIS

Deep abscesses in the neck are usually the result of infections in the oropharynx particularly of the teeth and tonsils Spread of infection within the compartments enclosed by the fascial planes in the neck usually results in a late diagnosis because clinical symptoms are usually not produced until pressure rises owing to pus within these compartments Serious spread along fascial planes and blood vessel sheaths may already have occurred to such vital areas as the mediastinum

Origin in Floor of Mouth

Ludwig s angina is a phlegmonous infection of the floor of the mouth, difficult of treatment and with a high morbidity rate The cause in most cases is an infection arising through the buccal mucosa Although many organisms have been involved, including the common pathogens the presence of anaerobes probably is important. A cellulitis and finally a phlegmonous abscess is formed between the floor of the mouth and the mylohyoid muscle since the latter is attached both to the mandible and to the hyoid bone it forms a closed compartment beneath the tongue both above and below the geniohyoid muscle The diagnosis of this infection is made on the presence of pain and tenderness in the mouth and the floor of the mouth and on painful swallowing The tongue and the structures of the floor of the mouth may be swollen Before the *diagnosis is made and treatment carried out* there may already be lymphatic spread to the larynx and even down into the lungs. Because of absorption of toxic materials these patients are usually very ill. The treatment of this deep phlegmonous abscess is adequately extensive incision into the suprahyoid area of the neck. One must not wait for suppuration to appear Systemic biotherapy is adjunctive and tracheostomy may be necessary

The most important deep abscess is that of the *retropharyngeal space* essentially from infections of the pharyngomaxillary fossa. Suppuration in this space may be mistaken for less serious infections of the oropharyngeal cavity and may descend rapidly along the prevertebral fascia into the mediastinum Such abscesses must be drained by transoral incision before this catastrophe occurs *Peritonsillar abscess* (quinsy) is an abscess in back of and around the tonsil the result of infection in that lymphoid structure It can be drained by opening the space posterior to the tonsil and usually is much less dangerous than the retropharyngeal abscess.

Origin in Mediastinum

Mediastinitis may also cause swellings on either or both sides of the neck owing to extension of the infection along fascial planes and, indeed may first become manifest in that direction Mediastinitis is most commonly caused by (1) contamination from the esophagus or the trachea following surgical instrumentation or traumatic injuries and by perforation of these viscera as a result of disease most commonly carcinoma, and (2) infection of mediastinal and hilar lymph nodes

SALIVARY GLANDS

Tumors

Tumors may arise in any of the salivary glands, but the parotid is the gland most

frequently affected Such tumors may involve single cell types or may be mixed" they may be benign or malignant and frequently they present characteristics of both *Mixed tumors* constitute the greatest number of the neoplasms of the salivary glands and are made up of elements from more than one germ layer Although histologically they appear benign and do not metastasize regionally or remotely local recurrence is the usual sequel to incomplete initial removal They are usually slow-growing, and their course may extend over many years although they often become progressively more malignant with successive recurrences following surgical extirpation *Carcinoma* of the salivary glands may occur as it does in any other epithelial structure The seventh cranial nerve (facial) traverses the substance of the parotid gland and is usually involved in malignant tumors it may be injured in operations upon this gland Treatment is by wide excision including the facial nerve if necessary Benign cysts and adenomas of the salivary glands also occur and can usually be easily enucleated.

Infections

Infections of the salivary glands are of two main types (1) pyogenic inflammations and (2) epidemic parotitis *Pyogenic inflammation* is usually a postoperative complication as a result of oral desiccation and poor hygiene Infection ascends from the mouth through the main ducts of the glands (Stensen s duct in the case of the parotid, Wharton s duct in the submaxillary) Presenting symptoms are those of a local infection (pain, swelling tenderness) plus the systemic signs Treatment is by systemic biotherapy with incision and drainage if fluctuation occurs Infections of the salivary glands may also be secondary to blockage of their main ducts by *calculi* Treatment is removal of the calculus and provision for drainage into the mouth *Epidemic parotitis* (*mumps*) is a specific virus cellulitus. Treatment is nonsurgical.

VASCULAR AND PARAVASCULAR ENLARGEMENTS

Aneurysms

Arteriosclerotic and traumatic arterial aneurysms and post traumatic arteriovenous fistulas occur in the great vessels of the neck and at the thoracic inlet The subclavian artery and vein in the supraclavicular space and the carotid arteries in the neck are common sites for these lesions They are discussed in detail in Chapters 10 and 11

Carotid Body Tumors

Carotid body tumor is one of the group known as *paragangliomas* The carotid body is located at the bifurcation of the common carotid artery at the level of the sixth cervical vertebra These tumors arise from elements of the sympathetic nervous system and contain *chromaffin tissue* (They occur elsewhere in the body i e the adrenals, the appendix the cecum, and the intestinal tract and are there called *carcinoid* or *argentaffin tumors*)

This interesting group of neoplasms has been the subject of a great deal of recent work with important additions to our knowledge It is now recognized that the cells in these tumors secrete serotonin This is a hormone (5-hydroxytryptamine) belonging to the catecholamine group and usually is excitatory to the cerebral centers and mimics the sympathetic portions of the autonomic nervous system When secreted in excessive quantities by argentaffin tumors widespread effects are noted including hypertension, particularly of the pulmonary circuit cutaneous hyperemia or flushing, especially of the face peptic ulceration and eventually failure of the right side of the heart. The presence of these tumors is detected by blood assay of serotonin and the urinary excretion of its degradation product, 5-hydroxy 3-indoleacetic acid.

The diagnosis of carotid body tumor is made on the basis of the syndrome just described, plus the finding of a progressively enlarging mass just below the angle of the

jaw which usually is without local symptoms until pressure is produced on adjacent cervical viscera The pulsation of the carotid artery is usually transmitted by this mass Because of their intimate attachment to the carotid arteries these tumors are mobile laterally but not vertically and they tend to extend inwardly They must be differentiated from branchiogenic cysts which grow out wardly About one-third of carotid body tumors are malignant Ideal treatment is total resection of the tumor This has not always been advisable especially if the tumor is benign since total removal may require ligation of the common carotid artery which will cause cerebral ischemia and hemiplegia in about 25 per cent of cases With newer technics of reconstruction of arteries this complication can now be avoided and cures by the removal of carotid body tumors should be the rule

TUMORS OF THE MANDIBLE

Tumors of the mandible include (1) those which arise in bone and (2) those of dental origin Tumors of bone which may be detected as masses in or near the neck are (1) osteomas, (2) giant-cell tumors, and (3) malignant tumors (sarcomas) Tumors of dental origin are (1) adamantinoma, a tumor of the anlage of enamel forming cells, (2) dentigerous cyst, which usually contains a true epithelial lining and a partially or fully developed tooth, and (3) radicular cyst which usually follows the removal of a tooth and arises from a paradental epithelium *Epulis* is a benign giant-cell tumor of the alveolar and gingival margins it has many characteristics of granuloma. It frequently follows dental extraction.

SUMMARY OF PRINCIPLES

1 Differential diagnosis of masses of the neck involves diseases of many origins many organ systems and all germ layers

2 Masses of congenital origin are due to errors in embryologic development Branchial cysts and sinuses are most commonly residuals of the first and second branchial clefts and furrows the second cleft derivatives are much more common they are usually found in the lateral triangles of the neck Thyroglossal duct cyst and sinus and lingual goiter are developmental defects in the descent of the original thyroid anlage to its ultimate normal resting place they are always in the midline. Hemangiomas and lymphangiomas are developmental arrests of vascular tissue

3 The thyroid gland enters into the differential diagnosis of masses of the neck as diffuse swelling of the thyroid gland nodules of the thyroid gland and lateral aberrant thyroid tissue

4 Lymphosarcoma is a multicentric disease commonly detected in lymph nodes of the neck It is a true neoplasm

5 The malignant lymphomas are a group of diseases which have some characteristics of granulomas and some of neoplasia and they usually follow a malignant clinical course. The viral cause of malignant lymphomas is a possibility

6 Metastatic malignancy of the lymph nodes of the neck is common secondary to lesions of the head and neck and of the lungs and gastrointestinal tract

7 Masses in the be the result of acute or chronic infections.

Acute pyogenic adenitis is frequently secondary to common infections of the head and neck Deep abscesses of the fascial compartments of the neck may be due to infection in the retropharyngeal space the peritonsillar area and the floor of the mouth Mediastinitis due to esophageal or tracheal disease, may first present as an infection in the neck The most common cause of chronic cervical lymphadenitis is tuberculosis

8 Tumors of the salivary glands may be benign or malignant Mixed tumors of the parotid gland are histologically benign and do not metastasize widely or remotely but they recur locally Involvement of the facial nerve is found early in malignant disease of the parotid gland Infections of the parotid gland may be of the epidemic variety (mumps) which is non surgical or may be secondary to poor hygiene in the postoperative period. The latter may become suppurative and require drainage

9 Aneurysms and arteriovenous fistulas of the subclavian and carotid vessels are not uncommon

10 Tumors of the mandible with masses in the neck may be of osseous or of dental origin

11 All common connective tissue and skin lesions are found in the cervical skin

SUGGESTED READINGS

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LYALL, D Lateral Cervical Cysts, Sinuses, and Fistulas of Congenital Origin (a collective review) *Surg Gynec & Obst* 102:417 1956.

15

The Breast

I Surgical anatomy and physiology
II Malformations
  A Congenital
  B Acquired
III Infections
  A Abscess
  B Tuberculosis
  C Other
IV Benign tumors
  A Fibrocystic disease
  B Papillomas
  C Schimmelbusch s disease
  D Fibroadenomas
  E Injury with fat necrosis
  F Plasma cell mastitis
V Cancer
  A Etiology and incidence
  B Pathology (1) histology (2) spread
  C Diagnosis and differential diagnosis (1) symptoms (with methods of examination) (2) signs
  D Treatment and prognosis

SURGICAL ANATOMY AND PHYSIOLOGY

The human female breast is a most distinctive secondary sexual structure Besides its obvious function of lactation and nursing it occupies a prominent place in the esthetics of the feminine form and in the psychology of both sexes. Like the uterus, the mammary gland is in a state of continuous and rhythmic structural change which parallels the gonadal activity Its histologic pattern varies with age maturity the stage of the menstrual cycle pregnancy lactation and the menopause As in all organs undergoing frequent and rapid morphologic alterations, irregularities in these changes often lead to morbid states many of which constitute stages of a continuing pattern

The breast is of ectodermal origin beginning as an invagination of the skin which ultimately becomes the nipple It is, in essence, a modified sebaceous gland designed for a specific function In the newborn of both sexes there is often a hyperplasia of the ductal system in response to the maternal circulating estrogens, sometimes accompanied by the secretion of a small amount of cloudy fluid (known in earlier times as *witches milk*) Until the age of puberty in both sexes the breasts consist almost exclusively of ducts with very little acinous structure formation. The male breast remains in this condition normally throughout life At puberty in the female

there is a marked proliferation of the ductal system and the formation of lobules of acini which progresses to the adult stage

The adult female breast lies upon the pectoral fascia is hemispherical in anterior posterior projection and is somewhat conical from the lateral view It is attached to the anterior chest wall between the second and seventh ribs and may extend from the parasternal to the midaxillary line There is often an extension of breast tissue toward and sometimes into the axilla (the axillary tail of Spence) Occupying the original center of the gland is the nipple and its surrounding areola of pigmented tissue under which lie both circular and radial muscles, which cause the nipple to become erect, and the glands of Montgomery which lubricate the nipple during lactation The nipple and areola vary in color and size in different individuals and at different times in life in a given individual The nipple becomes markedly enlarged during the late stages of pregnancy and particularly during lactation. The areola likewise becomes enlarged and more deeply pigmented under the same circumstances

The adult female breast consists of twelve or more compartments of glandular (acinous) tissue each compartment separated by a fibrous sheath the *suspensory ligaments of Cooper* The glandular tissue of the breast is the manufacturing division It is composed of clusters of acini, which are drained by a ductule the smallest unit in the milk-collecting system Many small ductules are gathered into larger collecting ducts and the latter are gathered into main ducts in or immediately subjacent to the nipple upon whose surface the milk is discharged Dispersed throughout the breast tissue is a varying amount of fat.

The blood supply to the breast arises chiefly from the following sources (1) the thoracoacromial branch of the axillary artery (2) the lateral thoracic branch of the axillary (3) perforating vessels from the intercostal arteries, and (4) perforating branches from the internal mammary arteries The venous drainage of the breast follows the same pattern as the arterial supply

The lymphatic drainage pattern of the breast is important in surgery because it determines the pathways of lymphatic dissemination and influences the course treatment, and prognosis of breast tumors The rich lymphatic network originates in periductal and periacinous lymphatics which communicate with interlobar vessels as well as with lymphatics in the subcutaneous tissue which ramify on the surface of the breast and on the pectoral fascia. The principal lymphatic drainage of the breast is into the axillary chain, especially from the lateral quadrants of the breast. The superior quadrants also have direct drainage to the supraclavicular nodes, and the medial quadrants drain directly to the internal mammary chain within the mediastinum There are also less prominent pathways along the rectus sheath and through the chest wall into the falciform ligament of the liver Since lymphatic vessels anastomose freely it is not possible to assume that drainage from any particular quadrant of the breast drains only to a particular group of nodes Unfortunately neoplasms may spread from any given portion of the breast to any of the areas that drain it, including across the sternum to the opposite breast.

The breast is under the control of the glands of the endocrine system particularly the ovaries and pituitary and to some extent the adrenal. During that part of life when the ovaries are active the breast undergoes a cyclic (usually monthly) pattern of *hyperplasia* and *involution* Estrogens secreted by the ovaries are a stimulant to growth and development of ductal tissue Progesterone produced in the second half of the menstrual cycle stimulates the lobular acini to growth and hyperplasia At this time of the month should impregnation of the uterus not have occurred, the hyperplastic changes produced by these two ovarian hormones recede (the process of involution) When impregnation of the

uterus has occurred, the breast goes on to extreme hyperplasia of both ductal and glandular tissue In the puerperium a hormone (prolactin) is secreted by the pituitary and lactation takes place The continual process of hyperplasia and involution is responsible for a number of the benign mastopathies discussed later in this chapter After the menopause, there is atrophy of all tissues of the breast, including connective tissue

MALFORMATIONS

Congenital

Malformations in size and number of the breasts are common. *Polymastia* more commonly known as *supernumerary nipples and breasts* is seen frequently Supernumerary nipples are far more frequently seen than supernumerary breasts They occur along the nipple line, which extends from the midclavicle to the groin on either side, most commonly caudad to the main breasts, although occasionally a pair is seen cephalad to them It would seem that these represent a phylogenetic throwback since more primitive mammals have two rows of breasts rather than two breasts The most extreme congenital malformation *amastia* or total absence of the breasts, is very rare Almost as rare is the congenital absence of a nipple.

Acquired

Anomalies in size and symmetry are also frequently seen The most common acquired malformation is that of massive hypertrophy This is not infrequently encountered at the age of puberty in females with a growth of the breasts out of all proportion to body size Histologic examination of such an organ reveals true hypertrophy of all elements, presumably under hormonal influence and not well understood When hypertrophy occurs in the male breast, the histology is quite different The male hypertrophic breast (gynecomastia) is entirely a hypertrophy of tubular structures. This occurs at about the time of puberty and/or in the fifth and sixth decades of life The breast growth in the male will usually disappear within a year of its onset, but the possibility of malignant disease cannot be glibly dismissed as will be indicated later in this chapter There is also a growth of the male breast in extensive liver disease and in disturbances of the testes. This is probably due to the excessive amount of circulating estrogens in the blood stream because the diseased liver is unable to metabolize this hormone.

INFECTIONS

Abscess

Most abscesses of the breast are puerperal They may occur not only during lactation but during pregnancy as well The origin of these abscesses is essentially exogenous, in that the edematous, hyperemic breast with the moist nipple is an excellent nidus for infection In times when sore throats are common there is an increased incidence of puerperal breast abscesses The diagnosis and principles of management of abscesses of the breast are the same as for such acute infections anywhere in the body The infected breast usually begins with an excoriated nipple, an area of "caking" and the typical findings of a soft tissue abscess. *Therapy includes incision and dependent* surgical drainage when the abscess becomes fluctant identification of the invading organism and appropriate antibiotic therapy In drainage of the abscess one must be careful to open all the locules of the abscess cavity in order to provide adequate drainage The incision must be made in a radial direction to avoid division of adjacent ducts. Weaning of the baby from the breast and drying of the secretions are adjunctive measures

Tuberculosis

Tuberculosis of the breast is not common but it is seen in those groups in society

where pulmonary tuberculosis is prevalent Usually the tuberculous infection will result in a draining sinus of the breast commonly on or near the areola The diagnosis and principles of therapy are the same as for tuberculosis anywhere These include identification of the organism by the typical histologic changes that it produces proper drainage and the use of drugs effective against *Mycobacterium tuberculosis*

Other

Fungus disease of the breast is occasionally seen Most of these cases are actinomycotic and are associated with actinomycetic infection elsewhere most frequently in the lungs and the cartilage of the sternoclavicular junction

BENIGN TUMORS

More than one-half of all patients with the presenting complaint to the physician of a lump in the breast have *benign mastopathies* Most of these are a result of irregularities of the physiologic processes of hyperplasia and involution under the influence of the sex hormones. These changes take on some of the characteristics of benign tumors, and the delineation between hypoplasia and neoplasia is frequently a tenuous one. Since the differential diagnosis of each of these lesions includes carcinoma of the breast, the definitive diagnosis usually rests with histologic examination of material taken by surgical excision

Fibrocystic Disease

Fibrocystic disease of the breast (commonly called *chronic cystic mastitis*) is one of the most common disease states of the adult female It is a source of worry (cancer phobia) to the patient and a diagnostic problem to the surgeon. This mastopathy is a result of the cyclic activity of the ovarian sex hormones upon the breast and of the repetitive hyperplasia-involution cycle which they induce It may occur in any woman of the age in which the ovaries are active regardless of parity and regresses after the menopause It is sometimes difficult to draw a sharp line of demarcation between normal physiologic activity in the breasts and a pathologic degree of fibrocystic disease

The pathology of this condition consists of the characteristic diffuse interstitial fibrosis in which cysts of various sizes are seemingly trapped sprinkled amongst the fibrosis and cysts (blue dome cysts) are areas of epithelial hyperplasia and an infiltration of lymphocytes. The lymphocytic infiltration accounts for the misnomer "mastitis" Any one or more of the above mentioned characteristics may predominate in a given area. In addition to the large thin walled cysts with smooth, fibrotic walls, there may be multiple ductal dilatation containing papillary excrescences or small adenomas

The presenting symptoms are either the palpation by the patient of lumpy or ropy areas in the breast or a marked sensation of tenderness, particularly during the premenstrual phase of her period. The degree of *mastodynia* (breast pain) varies more with the patient s threshold to pain than with the degree of cystic involvement. Some women with a great deal of fibrocystic disease have no symptoms whatever On physical examination the findings may vary from that of a single lump to that of a diffuse *nodular* and *ropy* character of both breasts. The entire breast may present a discord sensation on palpation The masses will usually be found to be smaller after a menstrual period there is a diminution in tenderness at this time as well These lumps do not always feel cystic but may be very hard to the touch The lumps slide freely under the skin and are not attached to the underlying pectoral fascia.

If one were absolutely sure that the nodules palpated were indeed fibrocystic disease and therefore benign, no treatment would be necessary However since the innocence of any given lump can only be suspected and not proved by physical examination, excision for biopsy must be performed Aspiration of a cyst percutaneously

will prove the benignancy of that given cyst when fluid is obtained, but gives no information about the surrounding tissue When both breasts are totally involved with symptomatic fibrocystic disease with repeated periods of excessive tenderness and especially with a fear of cancer, bilateral simple mastectomy is occasionally necessary Whether fibrocystic disease of the breast is premalignant in character is moot. Some large series report that the incidence of cancer in breasts which previously have had fibrocystic disease is higher than in the general population Other series cast serious doubt on this point.

Papillomas

Hyperplasia of ducts may result in papilloma formation from the ductal epithelium These growths may arise in any size ducts, but they are more frequently seen in the terminal ducts near the nipple They may be single or multiple These lesions frequently defy localization by palpation or any other means and can be suspected only by the production of a serous sanguinous, or grumous discharge from the nipple Since papillomas may proceed to papillary carcinomas the treatment is excision for biopsy Occasionally an entire quadrant of the breast will have to be removed and the papilloma located by examination of the specimen Diagnostic x ray examination of the breast is feasible only after injection of a radiopaque material through the nipple in an attempt to visualize an intraductal papilloma. When the papilloma cannot be found in the presence of a serous or grumous discharge this is a worthwhile procedure When the discharge is bloody it is best to proceed directly to surgical biopsy

Schimmelbusch s Disease

Schimmelbusch described a mastopathy consisting of the combination of lesions described *in the above two sections, i e., the* association of cysts and fibrous tissue together with a marked preponderance of intraductal papillomas. The latter are frequently too numerous to count and may easily be extruded by pressure on the specimen removed for biopsy Some pathologists refer to this histologic picture as *sclerosing adenomatosis* or *adenosis* and occasionally such a breast lesion is said to be carcinoma. Because of the possibility of formation of papillary carcinoma from the very large number of ductal papillomas, simple mastectomy is most frequently the treatment of choice Unfortunately some clinics use the term *Schimmelbusch s disease* synonymously with fibrocystic disease

Fibroadenomas

Fibroadenomas the characteristic solitary lesion in the young female are ordinarily found only during the menstruating age These lesions are discrete movable usually nontender and most often single There is a marked overgrowth of ductal tissue obvious on histologic examination as well as an abundance of fibrous tissue When the *fibrous tissue predominates the term adenofibroma* is sometimes applied There are two histologic varieties recognized, the intracanalicular and the pericanalicular types. This lesion is the direct result of the stimulation by estrogens of ductal tissue Local excision of these tumors is all that is required Sarcomatous change of a fibroadenoma only rarely occurs and these large lesions are known as *cystosarcoma phyllodes* and may reach massive size before metastasizing When a fibroadenoma is found in the breast of a girl before puberty or in the breast of a woman after the menopause the diagnosis of a granulosa cell or other estrogen-producing tumor of the ovary must be considered

Injury with Fat Necrosis

Fat necrosis consequent upon a blow to the breast is not uncommon since the large breast is frequently a fat depot Within weeks after the occurrence of such an injury a hard lump may be felt at the site of trauma. Excision and biopsy of such a lump will reveal the mineral salts of saponified

fats liberated by the trauma It is, of course much safer to excise such a mass for biopsy than to assume that it is fat necrosis and not a true tumor

Plasma Cell Mastitis

Plasma cell mastitis is an infrequent lesion of the breast in which there is a palpable mass under or near the areola. *Duct ectasia* would probably be a better name for this disease since there is obvious dilatation of the ducts with atrophy of their walls this entire area may be surrounded with a large number of plasma cells, which seem to be a response to the irritation of the ducts by the lipid fraction of breast secretion Histologic examination after excision for biopsy is diagnostic and local excision is adequate treatment.

CANCER

Etiology and Incidence

Cancer of the breast accounts for about one-fourth of all cancers in women. The ultimate direct cause of this carcinoma, as elsewhere is unknown. However the incidence of carcinoma in the breast is high, just as it is in all structures which are repeatedly subject to hyperplasia and involution. *Cystic mastopathy mazoplasia* (change of one type of epithelium into another), and *dysplasia* (abnormal tissue formation) lead to the formation of tumors.

Of all female breast tumors that are examined, approximately one half are malignant. Cancer of the breast occurs one hundred times as often in the female as it does in the male Of all these cancers there are 100 carcinomas to 1 sarcoma. About 55 per cent of all malignant disease of the breast occurs before the menopause has passed Although the highest incidence is in the forty to forty nine age group advancing age itself is a factor because in the seventy five to seventy nine age group the incidence is ten times that of the thirty five to thirty nine age group In Negroes the average age incidence is 5 years earlier probably because of earlier sexual maturation The incidence of cancer of the breast is greater in single than in married females in nonparous than in parous females in those who do not nurse their babies and in those who have abnormal lactation It is believed by some, on the basis of experimental evidence that there is some factor in milk passed on from mother to daughter which may represent a hereditary factor in this malignancy (Bittner milk factor) One frequently finds a family history of cancer in the breast in a patient with this lesion Some pathologists are convinced that the incidence of carcinoma in women who have had benign tumors previously is four and one half times as great as in those women who have had no benign tumors. Others are not convinced of the validity of these figures In this regard one can easily become involved in a discussion of "predeterminism" are the benign tumors precursors of cancer or are they already malignancies that defy diagnosis by available methods? There is a unanimity of opinion that virginal hypertrophy and excessive development of breasts shortly after puberty ultimately leads to a greater incidence of cancer than in the general female population. Trauma has frequently been implicated as a causative agent, but it is extremely difficult to prove.

Pathology

The largest number of cancers of the breast occur in the upper outer quadrant They usually arise from ductal structures a smaller number arise from acinar structures The ducts are under the influence of and directly stimulated by estrogens which are known to be potentially carcinogenic at least experimentally The acini are under the influence of and are directly stimulated by progesterone, which is not known to be carcinogenic, even experimentally Regardless of the type of cancer cell there is a stimulating effect of the neoplasm upon surrounding fibrous tissue *desmoplasia* The most common tumor of the breast is that

which stimulates the most marked amount of desmoplasia, *scirrhous carcinoma* This lesion is usually irregular in shape and infiltrating in all directions and has a hard rubber feel. When it is cut by a knife, a gritty sandy sensation is experienced The surface made by cutting appears to have white and yellow dots and streaks with no distinct boundary between normal tissue and tumor These tumors are hard and are fixed to the surrounding tissue and even to the skin above and the fascia below the breast Cancers of the breast which excite the least amount of fibrous tissue are referred to as *carcinoma simplex* or *medullary carcinoma* they are much less common. *Papillary carcinomas* are an end result of intraductal papillomas Because these intraductal lesions can sometimes be squeezed out of the ducts on the cut specimen they are referred to as *comedo-carcinoma* Papillary carcinomas are most common in or relatively near the nipple and the large collecting ducts Gelatinous or mucin-producing adenocarcinomas are less common They grow slowly and metastasize late *Paget's disease* of the breast is a relatively uncommon lesion and is a ductal cancer of the breast associated with a desquamation of the skin of the nipple. The main tumor is usually found not far from the areola *Inflammatory carcinoma* of the breast is the name given to a rapidly spreading invasive tumor which early blocks all lymphatics in the breast and its skin and which in some ways superficially resembles an inflammatory disease

There are several modes of spread of tumor of the breast. *Local* and *regional* invasion by direct growth and extension to the skin and to the underlying pectoral fascia, as well as to Cooper s ligaments is characteristic. It is the fixation to the skin and to Cooper s ligaments that produces the characteristic dimpled appearance, to be described later and the attachment to the pectoral fascia below which fixes the breast upon the chest wall Extension to regional lymph nodes occurs via lymphatic channels. These are mainly to the axilla and supra-

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growth compared with previous examinations Upon occasion transillumination with a small light in a dark room is of value Granular tissue does not transmit light well fat somewhat better and cysts best of all *Puckering* of the skin and *retraction* of the nipple is occasionally brought out when the patient assumes a position in which the breasts are pendant i e bending forward *Dimpling* of the skin above a tumor is a direct result of the fixation of that tumor to the skin by involvement of the cutaneous lymphatics *Retraction of the nipple* is a direct result of extension of the tumor into one or more of Cooper s ligaments *Bleeding* from the nipple must be assumed to be due to an intraductal papilloma or papillary carcinoma, until proved otherwise by excisional biopsy When the lymphatics in and under the skin of a relatively large area of the breast are involved by cancer cells the fixation of the skin glands to the underlying tissue gives the skin a characteristic appearance said to resemble the peel of an orange (*peau d'orange*) this appearance is also referred to as *pigskin* This serious finding represents not only fixation to but edema of the skin Dimpling retraction and pigskin appearance are all late signs When inflammatory carcinoma exists the entire breast is so obviously involved and fixed that the term *carcinoma en cuirasse* (literally breast plate or body armor) has been applied Any eruption of the nipple or its surrounding area, especially if chronic is suspect as part of Paget s disease of the nipple implying an underlying malignant tumor The presence of tumors in the skin of the breast when a tumor exists in the depth of the breasts is an ominous sign and usually indicates cutaneous metastases *Ulceration* of the skin of the breast by a tumor is usually a very late sign This occurs in tumors which grow very large before metastasizing, such as the medullary carcinomas Palpation of the axilla for the presence size and mobility of lymph nodes is very important in both diagnosis and prognosis.

The diagnosis of cancer of the breast must be entertained whenever a mass is palpated in the breast *Biopsy* of lesser lesions must be made in order to exclude the diagnosis of carcinoma from the following list of lesions (1) fibrocystic disease (2) fibroadenoma (3) Schimmelbusch s disease (4) sclerosing adenomatosis, and (5) fat necrosis.

Treatment and Prognosis

Whenever a patient is brought to the operating room for excisional biopsy of a breast tumor that patient must be mentally prepared in advance for the possibility that a radical mastectomy may have to be performed The operating room must be prepared for radical mastectomy in every case of excision for biopsy When cancer is found in the surgical specimen the biopsy incision should be closed If a radical mastectomy is to be performed the skin should be prepared again and a different set of instruments used for the larger operation This precaution is worthwhile in order to avoid *seeding* of the wound with cancer cells, which have been shown to be present and viable by tissue culture on instruments used for biopsy The general principle of treatment of cancer of the breast is the same as for treatment of cancer of any other organ and includes the removal of the diseased organ and as much of its lymphatic bearing adjacent tissue as is surgically practical This operation is known as radical mastectomy It is considered by many to be a misnomer since the internal mammary chain of lymph nodes is ordinarily not included in this surgical procedure The operation as originally described by Halsted and by Meyer includes excision of the breast, the pectoral musculature sufficient skin and the axillary contents in an en bloc dissection designed to minimize possibilities of cutting across cancer tissue

There are two general departures from this conventional surgical attack The first is that of McWhirter which consists of simple mastectomy followed by intensive postoperative irradiation the other the superradical procedure popularized by Urban and

which stimulates the most marked amount of desmoplasia, *scirrhous carcinoma* This lesion is usually irregular in shape and infiltrating in all directions and has a "hard rubber" feel When it is cut by a knife a gritty sandy sensation is experienced The surface made by cutting appears to have white and yellow dots and streaks with no distinct boundary between normal tissue and tumor These tumors are hard and are fixed to the surrounding tissue and even to the skin above and the fascia below the breast. Cancers of the breast which excite the least amount of fibrous tissue are referred to as *carcinoma simplex* or *medullary carcinoma* they are much less common *Papillary carcinomas* are an end result of intraductal papillomas Because these intraductal lesions can sometimes be squeezed out of the ducts on the cut specimen they are referred to as *comedo-carcinoma* Papillary carcinomas are most common in or relatively near the nipple and the large collecting ducts Gelatinous or mucin-producing adenocarcinomas are less common They grow slowly and metastasize late *Paget's disease* of the breast is a relatively uncommon lesion and is a ductal cancer of the breast associated with a desquamation of the skin of the nipple The main tumor is usually found not far from the areola *Inflammatory carcinoma* of the breast is the name given to a rapidly spreading invasive tumor which early blocks all lymphatics in the breast and its skin and which in some ways superficially resembles an inflammatory disease

There are several modes of spread of tumor of the breast *Local* and *regional* invasion by direct growth and extension to the skin and to the underlying pectoral fascia as well as to Cooper's ligaments, is characteristic. It is the fixation to the skin and to Cooper's ligaments that produces the characteristic dimpled appearance to be described later and the attachment to the pectoral fascia below which fixes the breast upon the chest wall Extension to regional lymph nodes occurs via lymphatic channels These are mainly to the axilla and supraclavicular area in lesions of the outer quadrants of the breast and to the internal mammary chain within the mediastinum in medial quadrant lesions. Distant metastases occur by hematogenous spread through *venous embolization* These tumor emboli appear to grow most readily in the lungs and in the skeleton Lymphatic and hematogenous spread to the liver are also common, and hematogenous embolization to the brain is not uncommon In recent years cancer cells have been found in the circulating blood in the presence of cancer of many organs It remains for the future to refine the ability to detect circulating cancer cells and to destroy them chemically Osseous involvement usually is at first osteoclastic but osteoblastic activity may occur at the periphery of the metastic lesions.

Diagnosis and Differential Diagnosis

Diagnosis of cancer of the breast may be easy and obvious or at times, very difficult. In any event the ultimate diagnosis must be made by wide excision and microscopic examination The symptom most commonly presented by the patient is the palpation of a mass in the breast while bathing Occasionally no symptoms whatever are presented, and the tumor is discovered when the examining physician palpates a mass.

The breasts must be inspected from every angle Any asymmetry must be taken note of (although there is usually a disparity in size between the right and left breasts in most patients) and most particularly the skin must be observed for changes The breasts must be palpated between the fingers of both hands as well as against the chest wall Fixation to the pectoral fascia or to the skin is noted and the presence of any nodes in the supraclavicular area or the axilla is sought for A note is made of the size of any lesion, its *contour* (whether smooth rough or lobulated) its *discreteness* (whether it is circumscribed encapsulated or ill-defined) its *consistency* (soft hard, resilient or fluctuant) its *mobility* (whether free or attached) and its *rate of*

growth compared with previous examinations. Upon occasion transillumination with a small light in a dark room is of value. Granular tissue does not transmit light well, fat somewhat better, and cysts best of all. *Puckering* of the skin and *retraction* of the nipple is occasionally brought out when the patient assumes a position in which the breasts are pendant, i.e., bending forward. *Dimpling* of the skin above a tumor is a direct result of the fixation of that tumor to the skin by involvement of the cutaneous lymphatics. *Retraction of the nipple* is a direct result of extension of the tumor into one or more of Cooper's ligaments. *Bleeding* from the nipple must be assumed to be due to an intraductal papilloma or papillary carcinoma until proved otherwise by excisional biopsy. When the lymphatics in and under the skin of a relatively large area of the breast are involved by cancer cells, the fixation of the skin glands to the underlying tissue gives the skin a characteristic appearance said to resemble the peel of an orange (*peau d'orange*); this appearance is also referred to as *pigskin*. This serious finding represents not only fixation to but edema of the skin. Dimpling, retraction, and pigskin appearance are all late signs. When inflammatory carcinoma exists, the entire breast is so obviously involved and fixed that the term *carcinoma en cuirasse* (literally, breast plate or body armor) has been applied. Any eruption of the nipple or its surrounding area, especially if chronic, is suspect as part of Paget's disease of the nipple, implying an underlying malignant tumor. The presence of tumors in the skin of the breast when a tumor exists in the depth of the breasts is an ominous sign and usually indicates cutaneous metastases. *Ulceration* of the skin of the breast by a tumor is usually a very late sign. This occurs in tumors which grow very large before metastasizing, such as the medullary carcinomas. Palpation of the axilla for the presence, size, and mobility of lymph nodes is very important in both diagnosis and prognosis.

The diagnosis of cancer of the breast must be entertained whenever a mass is palpated in the breast. *Biopsy* of lesser lesions must be made in order to exclude the diagnosis of carcinoma from the following list of lesions: (1) fibrocystic disease, (2) fibroadenoma, (3) Schimmelbusch's disease, (4) sclerosing adenomatosis, and (5) fat necrosis.

Treatment and Prognosis

Whenever a patient is brought to the operating room for excisional biopsy of a breast tumor, that patient must be mentally prepared in advance for the possibility that a radical mastectomy may have to be performed. The operating room must be prepared for radical mastectomy in every case of excision for biopsy. When cancer is found in the surgical specimen, the biopsy incision should be closed. If a radical mastectomy is to be performed, the skin should be prepared again and a different set of instruments used for the larger operation. This precaution is worthwhile in order to avoid *seeding* of the wound with cancer cells, which have been shown to be present and viable by tissue culture on instruments used for biopsy. The general principle of treatment of cancer of the breast is the same as for treatment of cancer of any other organ and includes the removal of the diseased organ and as much of its lymphatic bearing adjacent tissue as is surgically practical. This operation is known as radical mastectomy. It is considered by many to be a misnomer since the internal mammary chain of lymph nodes is ordinarily not included in this surgical procedure. The operation, as originally described by Halsted and by Meyer, includes excision of the breast, the pectoral musculature, sufficient skin, and the axillary contents in an en bloc dissection designed to minimize possibilities of cutting across cancer tissue.

There are two general departures from this conventional surgical attack. The first is that of McWhirter, which consists of simple mastectomy followed by intensive postoperative irradiation; the other, the superradical procedure popularized by Urban and

be evaluated on an individual basis Estrogens are most useful in causing the temporary disappearance of and relief from pain in soft tissue metastases Under no conditions should the estrogens be used before the age of menopause The adrenal corticosteroids sometimes retard the growth of metastases and diminish pain therefrom The value of adrenalectomy and of hypophysectomy in the treatment of metastatic tumors of cancer of the breast is not yet firmly established

It might be expected that cancer of the breast an accessible and visible organ would carry the greatest long term non recurrence rate Yet such is not the case partly because the involved breast is not seen early enough by the surgeon and partly because of the difficulty in making a diagnosis in many instances Some cases are seen for the first time at such an advanced stage that no reasonable prolongation of life may be expected with the use of the conventional radical mastectomy It has been found from experience in many quarters that there are certain criteria of inoperability These include (1) edema of more than one third of the skin over the breast (2) satellite tumors of skin over the breast (3) parasternal or intercostal chest wall tumor nodules and supraclavicular nodes, (4) edema of the arm preoperatively (5) inflammatory carcinoma usually occurring during pregnancy or lactation and (6) any two of the following (*a*) ulceration of the skin (*b*) edema of one-third of the skin (*c*) fixation to the chest wall (*d*) axillary nodes greater than 2.5 cm in transverse diameter if proved by biopsy and (*e*) fixation of involved nodes to the skin Even if there is an unequivocal criterion of inoperability simple mastectomy may be worthwhile particularly if there is a large breast tumor which is potentially ulcerating and productive of foul discharge

In general the prognosis of cancer of the breast is much poorer under the age of forty than over this age Similarly a generalization can be made that the shorter the duration of symptoms the greater prolongation of life may be expected Probably the greatest prognostic significance relates to whether the tumor is infiltrating or circumscribed the former having the poorest prognosis the latter the best With regard to location of the tumor within the breast the poorest prognosis is for those tumors in the medial quadrants since metastases to the internal mammary lymph node chain and possibly from there along the falciform ligament to the liver have already occurred Blood vessel invasion makes for a dismal prognosis A malignant tumor of the breast during pregnancy and lactation is almost uniformly fatal It is fairly well agreed that an incomplete operation compromises the ultimate result

When a breast is removed with the histologic diagnosis of cancer the surgeon may expect to find a subsequent cancer in the other in 3 to 8 per cent of the cases. Although some believe that this is a metastatic involvement via lymphatics in the skin and across the sternum the probability is that it is a separate and distinct malignant tumor It is therefore not unreasonable to recommend simple mastectomy of the contralateral side when a malignant tumor of the breast is found.

SUMMARY OF PRINCIPLES

1 The breast is a modified ectodermal (sebaceous) gland designed for the specific function of lactation

2 Many gland clusters and the ductules which lead to the main collecting ducts are compartmentalized into 12 or more divisions divided by the suspensory ligaments of Cooper The nipple collects all of the main ducts

3 The rich lymphatic network of the breast is important in the treatment and prognosis of cancer of this organ.

4 The breast is under control of the glands of the endocrine system particularly the ovaries and pituitary and to some extent the adrenal The ducts are under the influence of circulating estrogens the acini under the influence of progesterone Under the influence of these two hormones, the breast undergoes a cyclic alternation of hyperplasia and involution throughout the period of life when the ovaries are active

5 Malformations of the breast include polymastia amastia, and the acquired anomalous massive virginal hypertrophy in the female Male hypertrophy of the breasts (gynecomastia) is entirely of tubular structures.

6 The most common benign mastopathy is fibrocystic disease a result of the repetitive hyperplasia involution cycle

7 Intraductal papillomas are a result of extreme hyperplasia of breast ducts

8 Schimmelbusch s disease is the occurrence of extreme intraductal papillomatosis with fibrocystic disease

9 Fibroadenoma is the characteristic solitary lesion of the young female and is the result of the stimulation of ductal tissue by estrogens.

10 Plasma cell mastitis is a mastopathy characterized by duct ectasia surrounded by plasma cells

11 Cancer of the breast accounts for about one fourth of all cancers in women and is most common at about the time of the menopause Its cause is not completely understood some predisposing factors are presented in the chapter

12 Most breast cancers arise from ductal tissue and a smaller number from acinar tissue

13 Desmoplasia is the excitation by a neoplasm of fibrous tissue surrounding it.

14 The most common breast cancer is the scirrhous carcinoma less common are carcinoma simplex or medullary carcinoma papillary carcinoma or comedo-carcinoma gelatinous adenocarcinoma Paget s disease and inflammatory carcinoma

15 Cancer of the breast spreads by local and regional invasion by direct growth by extension along lymphatic channels and via the hematogenous route

16 Examination of the breast includes inspection and palpation and only occasionally aspiration and x ray examination with radiopaque material The lump is examined for contour discreteness consistency mobility and rate of growth as well as its ability to transmit light Puckering of the skin retraction of the nipple bleeding from the nipple ulceration of the skin and fixation of the breast to the skin or to the pectoral fascia are sought for

17 The only definitive diagnosis of a tumor of the breast is by excision and histologic examination (biopsy)

18 Treatment of cancer of the breast is essentially an en bloc radical mastectomy

19 The prognosis following radical mastectomy is much poorer when there is malignant involvement of the axillary lymph nodes

20 Adjuvants in the treatment of cancer of the breast are irradiation and the use of androgens and estrogens the former for osseous metastases the latter for soft tissue spread

21 Criteria of inoperability are the presence of two or more of the following (*a*) edema of skin (*b*) cutaneous metastases over the breast (*c*) supraclavicular metastases (*d*) edema of the arm (*e*) inflammatory carcinoma (*f*) ulceration of the skin (*g*) fixation to chest wall and (*h*) axillary nodes greater than 2 5 cm in transverse diameter

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16

Introduction to Surgery of Pulmonary and Bronchial Diseases; Pulmonary Atelectasis

I **Anatomy and physiology**
II **Methods of study and diagnosis**
  A History and physical examination The origin of symptoms and signs
  B Sputum (1) bacteriology (2) cytology
  C Bronchoscopy (1) diagnostic (2) therapeutic
  D Roentgenography (1) plain (2) stereo and tomography (3) bronchography
III **Pulmonary atelectasis**
  A Definition
  B Etiology (1) extrinsic pressure (2) postoperative (intrinsic) (*a*) predisposing factors (*b*) precipitating factors
  C Pathology
  D Diagnosis
  E Therapy (1) prophylactic (2) definitive

ANATOMY AND PHYSIOLOGY

The respiratory system performs the dynamic function of bringing oxygen from the external environment and transmitting it to the vascular system Transportation of the oxygen from the outside to the alveolar wall is known as *ventilation* It is accomplished by a complex regulatory mechanism which is discussed in detail in Chapter 17 The air conduction system beyond the oropharyngolaryngeal pathways (upper respiratory tract) is the tracheobronchial tree (lower respiratory tract) The trachea is a midline tubular structure composed of cartilaginous rings and a soft dorsal wall it extends from the level of the sixth cervical vertebra (the larynx) to its carina at the level of the second intercostal spaces (angle of Lewis) retrosternally where it bifurcates into its two main divisions the right and left main bronchi (Fig. 23) The bronchi further are subdivided into lobar segmental and subsegmental bronchioles Although these subdivisions are subject to numerous variations, in general they follow a relatively constant pattern Ordinarily there are three lobes of the right lung and two of the left The lingula of the upper lobe is sometimes considered the analogue of the middle lobe of the right lung A knowledge of the topographic anatomy with relation to the bronchopulmonary segments of the various lobes is necessary in roentgenographic, bronchoscopic and

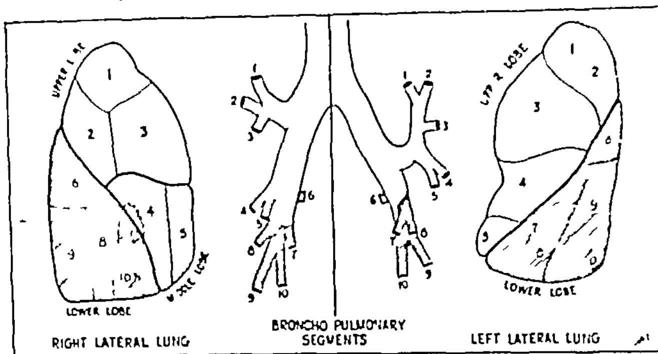


FIG 23 The bronchopulmonary segments (Jackson Huber) The upper lobes are in white the middle lobe of the right lung is stippled the lower lobes are striated. The lingula of the upper lobe of the left lung is the analogue of the right middle lobe Numbers indicate pulmonary segments and their corresponding bronchi

bronchographic diagnosis. The tracheobronchial tree is lined by a ciliated transitional epithelium mucus producing in type

Respiratory function is accomplished at the alveolar level by transport of gases in both directions across its epithelium into vascular capillaries Normal pulmonary function is the consummate ability of the respiratory system to maintain normal oxygen and carbon dioxide relationships in the blood at a normal respiratory rate under conditions of pressure and volume changes in the thorax which leave the patient virtually unaware of his breathing Respiratory dysfunction is a disturbance in any of the mechanisms involved in normal respiration. Respiratory insufficiency is a dysfunction which is productive of symptoms. Pulmonary function is measured by simple spirometry and bronchospirometry and by the ability of the respiratory system to adapt to an increased work load. Where surgical therapy may involve a large part of the patient's lung it is important to know the functional capacity of the residual lung before operative therapy is carried out.

METHODS OF STUDY AND DIAGNOSIS

History and Physical Examination. The Origin of Symptoms and Signs

The presenting symptomatology in diseases of the chest frequently gives an indication of the nature of the disease process The cardinal symptoms of pulmonary disease are *cough pain hemoptysis* and *dyspnea* Cough is produced by irritation of respiratory epithelium and is therefore indicative of some form of endobronchial disease, whatever else may be present. Pain is usually indicative of pleural irritation and, therefore most commonly of subpleural pathology Hemoptysis, the coughing up of blood, indicates the escape of blood from the vascular tree this may be at the alveolar level or may be visible per endoscopy in the bronchial tree Dyspnea (shortness of breath) is complex and sometimes very difficult of evaluation It may be due to ventilatory *insufficiency* such as obstruction of a bronchus it may be due to pure respiratory insufficiency such as obtains when there is

not enough alveolar wall exposed to inspired air (pneumonia) it may be due to the combined complex ventilatory and respiratory insufficiency of a degenerative process with loss of pulmonary elasticity such as is found in emphysema. Dyspnea may also be produced on the basis of overloading of the pulmonary vascular circuit, acutely or chronically as is experienced in congestive heart failure

Physical examination in diseases of the chest is not complete without a detailed examination of the neck for adenopathy abnormal pulsations, and venous distention Determination of oxygen saturation levels in the blood and the search for evidence of respiratory acidosis or alkalosis frequently are complementary in the diagnosis of surgical pulmonary diseases Systemic signs of suppurative disease are commonly present and must be a part of routine evaluation.

Sputum

Examination of the sputum is the first thought in laboratory diagnosis of pulmonary diseases. Of primary importance is the search for the organism of tuberculosis This is true even in this day when great strides have been made in the control of the disease Smears of sputum must be repeatedly examined by properly trained personnel before the presence of tuberculosis can be excluded. When indicated, search is made for other pathogens, including fungi Cultures and guinea pig inoculation studies complete the search for the *Mycobacterium tuberculosis* The examination of sputum for exfoliated cells is a most valuable contribution to diagnostic methods. When positive *exfoliative cytology* is more reliable in diagnosis of malignant disease than is roentgenographic interpretation

Bronchoscopy

Bronchoscopic examination has become an integral part of diagnosis of pulmonary disease Not only may the endobronchial tree be visualized but sputum may be taken from the orifices of the segmental bronchi for examination and pieces of tissue may be removed for biopsy In preoperative preparation for pulmonary resection in tuberculosis, bronchoscopy is of extreme importance in assessing the presence absence or degree of endobronchial involvement

Roentgenography

Survey films of the chest should always include posteroanterior lateral, and oblique views because some shadows within the lung fields are not seen on the posteroanterior view their densities being overshadowed by the cardiac silhouette Stereoscopic films frequently help to define the nature and position of space-occupying lesions Tomograms, or laminograms, are frequently of critical value in defining the position, size and shape of a mass and in determining whether it contains a cavity or not Serial body section radiograms are extremely helpful in assessing the benefit of therapy employed. At the time of bronchoscopy a soft catheter may be left in the tracheobronchial tree for the instillation of radiopaque materials and the visualization both fluoroscopically and on film, of the bronchial tree This is of particular value before resection for bronchiectasis is undertaken. Bronchography should be accomplished with materials which are easily expelled from the alveoli This is not a totally innocuous procedure and its use should be reserved for situations which cannot be resolved adequately by the usual diagnostic methods.

PULMONARY ATELECTASIS

Definition

The term *pulmonary atelectasis* (literally incomplete expansion) means the absence of air in the pulmonary alveoli This condition varies greatly in extent and distribution. It may be localized or diffuse patchy or massive massive atelectasis indicates that one or more pulmonary lobes are functionless.

Etiology

Atelectasis is generally due to bronchial obstruction which may be caused by extrinsic compression or by intrinsic occlusion. Obstruction of the bronchial lumen by compression from without usually results from enlarged lymph nodes, tumors and less frequently vascular anomalies in the very young. *Postoperative pulmonary atelectasis* is a frequent surgical complication and is usually due to a diffuse bronchiolar obstruction. It is attributable to a combination of two factors: (1) thickened secretions which cannot be evacuated by normal ciliary action or coughed out by the ordinary tussive effort and (2) the exaggerated resilience of the lung tissue (spasm) in response to inhalant anesthetics and postoperative pain. Factors which predispose to postoperative pulmonary atelectasis are pre-existent allergic disease and suppurative bronchial infections. There are two main precipitating factors in postoperative pulmonary atelectasis: (1) *Diminished respiratory excursion* in the early postoperative period is frequent and characteristic. Pain in the chest and upper abdomen following thoracic or abdominal operations limits the respiratory excursion; this shallow breathing does not adequately expand the lung. Accumulating secretions then block the bronchioles and smaller bronchi and the alveoli which they serve become airless. (2) The elevation of the diaphragm by abdominal distention (i.e. ileus) from any cause and the use of abdominal binders are conducive to the development of atelectasis, especially of the compressed lower lobes.

Pathology

When an appreciable portion of a lung is airless, there is a decrease in size of the ipsilateral hemithorax since the pulmonary volume is smaller. Accordingly the mediastinum is shifted toward the side of atelectasis. This is further aggravated by the compensatory emphysema of the contralateral lung which responds with increased inspiratory expansion to maintain normal gaseous exchange. The pathologic sequelae of atelectasis are (1) pneumonitis in the early postatelectatic period and (2) fibrosis and intractable emphysema in subsequent weeks and months.

Diagnosis

The symptoms of pulmonary atelectasis in the early postoperative period are moist cough and wheezing respiration. Later when infection supervenes there will be fever, toxemia and the physical signs of pneumonitis. Physical examination reveals shallow respiratory excursion, tachypnea (rapid respiration), rales on auscultation, compensatory tachycardia and frequently fever. Massive atelectasis from collapse of one or more pulmonary lobes is an acute and alarming syndrome with dyspnea, cyanosis, syncope and extreme tachycardia, a picture closely resembling pulmonary embolism. Dullness may be elicited over the atelectatic area after about 48 hr. and at that time a triangular shadow corresponding to the atelectatic segment may be seen roentgenologically. In massive pulmonary collapse mediastinal shift and elevation of the diaphragm may be demonstrated by physical and radiographic examination immediately after the catastrophe has occurred. The reason for the lapse of time before roentgen-ray evidence is positive is the fact that time is required for the air trapped in the alveoli to be absorbed.

Therapy

The therapy of pulmonary atelectasis begins logically with prophylactic measures in the preoperative period. Careful attention should be paid to pre-existing pulmonary disease, allergic or inflammatory. History of allergies should be elicited and all precautions used to avoid allergenic factors. Asthmatic breathing demands diagnosis and therapy prior to elective surgery. It may be due to the pulmonary edema of cardiac decompensation, to allergic swelling of the bronchial mucosa, or to the presence of active

suppurative disease Cardiac compensation should be attained, and antihistaminics and antibiotics should be used Inhalant (aerosol) therapy is appropriate at this time as well as after atelectasis has occurred. In the early postoperative period the patient should be instructed with regard to the importance of deep breathing as well as the necessity for coughing even if some pain is experienced. The use of high abdominal binders should be discouraged. Prolonged paralysis of respiratory musculature during long anesthesia is not advisable Local anesthetic block of the appropriate intercostal nerves *is most beneficial in preventing pain along* the area innervated by the intercostal nerves, in thoracic and upper abdominal incisions.

Once the diagnosis of atelectasis has been made several avenues of approach for its correction are available Since atelectasis is due to the combination of thickened mucus in the small bronchioles and to the spasm of these small ducts agents which liquefy the mucus and which dilate the bronchi are indicated. Both *wetting agents* and *bronchodilators* are advantageously used by the inhalation (aerosol) technic Bronchodilators, in general, are of the norepinephrine group wetting agents, in general, are those which lower the surface tension of the highly viscid bronchial mucus. Some medicaments such as the iodides and the guaiacol sulfonates, are useful when taken by mouth. Inhalations of carbon dioxide or the use of carbon dioxide–oxygen mixtures (3 to 7 per cent CO_2) are useful in the production of deep inhalations. *Tracheobronchial toilet* the introduction of a soft catheter into the trachea down (at least) to the carina, is useful for the purpose of producing endobronchial irritation and stimulating cough reflex It can be performed after minimal training, by the floor nurse Suction applied to the catheter occasionally dislodges casts of the bronchi More frequently the catheter induces a tussive effort which dislodges mucus in the very small bronchioles which cannot be reached directly by any catheter Occasionally especially in massive atelectasis formal bronchoscopy may be necessary to aspirate a large plug of mucus In the vast majority of cases however combination of aerosol therapy and bedside tracheobronchial toilet are adequate to overcome the obstructive atelectasis. During this time antibiotics are properly useful, since pneumonitis is the usual sequel to the inactive or atelectatic lung

SUMMARY OF PRINCIPLES

1 Pulmonary atelectasis is the absence of air in a patch a segment or a lobe of the lung

2 The cause of atelectasis may be obstruction from outside the bronchus or occlusion of the lumen from within

3 Postoperative atelectasis is occlusion of the bronchus due to (*a*) thick secretions which cannot be coughed out or expelled and (*b*) an exaggeration of the normal resilience of the small bronchioles (spasm)

4 Predisposing causes to postoperative atelectasis are pre-existing pulmonary diseases such as allergy and infectious bronchial disease, and passive hyperemia of congestive heart failure.

5 Precipitating causes for postoperative atelectasis are the decreased respiratory excursion consequent upon the experience of pain due to thoracic or upper abdominal incisions Elevation of the diaphragm due to abdominal distention or the use of abdominal binders is conducive to atelectasis of the lower lobes

6 The mediastinum is shifted toward the side of atelectasis The opposite lung undergoes compensatory emphysema

7 The diagnosis of atelectasis is based on the findings of moist cough rales and wheezes shallow respiration tachypnea and tachycardia fever and the typical x ray findings

8 Prophylactic treatment of postoperative atelectasis begins in the preoperative period with the evaluation and treatment of pre-existing pulmonary conditions be they allergic or infectious Prophylaxis further includes the encouragement of deep breathing and of coughing the avoidance of high abdominal binders the avoidance of long respiratory muscular paralysis and most important of all the anesthetization locally of intercostal nerves carrying incisional pain

9 The definitive therapy of postoperative pulmonary atelectasis includes the use of bronchodilators wetting agents carbon dioxide inhalation antibiotics tracheobronchial toilet and bronchoscopy

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17

Wounds of the Thorax and Surgery of the Pleural Space

I Physiology of respiration
  A Ventilatory and respiratory phases
II Pathologic physiology in trauma and disease
III. Trauma to the thorax
  A Types—blunt, penetrating and perforating
  B Clinical findings—diagnosis pathology and treatment (1) fracture of ribs (2) flail chest (3) hemothorax (4) pneumothorax (5) tension pneumothorax (6) pneumomediastinum and tension pneumomediastinum (7) sucking wounds (8) vascular compression syndrome (9) cardiac injuries (10) combined thoraco-abdominal injuries
IV Pleural effusion
  A Types (1) serous and sanguinous (2) purulent (3) chylous
  B Clinical causes (1) trauma and cardiac decompensation (2) secondary to lung pathology (a) pneumonia (b) abscess (c) tuberculosis (d) carcinoma (3) secondary to lymphatic obstruction and diaphragmatic and subdiaphragmatic pathology
  C Treatment of effusions
  D Course and sequelae (1) pleural thickening (2) empyema (3) fibrothorax
  E Treatment of empyema and fibrothorax (1) closed (2) enzymatic debridement and open operation (3) fibrothorax and decortication

PHYSIOLOGY OF RESPIRATION

Ventilatory and Respiratory Phases

The logical and successful treatment of wounds and diseases of the thorax requires accurate knowledge of the mechanisms involved in normal respiration and the pathologic physiology of trauma and disease

Normal respiration is dependent upon the anatomic integrity of the thoracic cage with its two pleural compartments and the intervening mediastinum and upon the complex neurologic and chemical control of the respiratory cycle as well as the functional integrity of the alveolar respiratory comment.

Ventilation is accomplished by the motions of inspiration and expiration (Fig 24) The inspiratory effort is made by muscular action under neurologic control By action of the intercostal muscles and the diaphragm the thoracic cage increases the lungs follow the thoracic wall outward and

thus their total volume is increased A negative pressure of about 10 to 15 cm H_2O is thereby produced in the pleural space (from a resting negative pressure of 2 to 4 cm H_2O) Air is then drawn into the lungs through the trachea (The space between the visceral and parietal pleurae is a potential space only the visceral and parietal pleurae being in apposition)*

The expiratory phase of the ventilatory effort is accomplished by relaxation of the muscles of respiration and by the elastic recoil of the lungs toward their preinspiratory size This recoil is made possible by the presence of a great amount of elastic tissue within the parenchyma of the lung Any impairment in the functions of inhalation and exhalation constitutes a derangement of the ventilatory phase of respiration The true *respiratory* phase of respiration the interchange of gases across the alveolar membrane is dependent upon the anatomic and functional integrity of the alveolus (Figs 25 to 30)

* The increase in negative pressure within the thorax during the inspiratory act is an *essential* part of the venous heart the mechanism for return of blood from the lower extremities and the abdominal viscera to the right side of the heart.

PATHOLOGIC PHYSIOLOGY IN TRAUMA AND DISEASE

The presence of fluid or air in the pleural space in sufficient amounts may disturb the dynamics of the respiratory cycle *Pneumothorax* (air in the pleural space) prevents the lung from following the thoracic wall outward during inspiration and thus reduces the ventilatory efficiency of that lung Such accumulation of air may result from rupture of alveoli by disease or trauma or may be introduced from the outside when the integrity of the thoracic wall is breached. If there is a persistent opening in the thoracic wall which permits the continuous flow of air between the outside and the pleural space intrapleural pressure *remains* at atmospheric levels Under such conditions the lung on that side cannot expand. This situation obtains when an accidental wound of the thoracic cage occurs or during thoracotomy the purposeful opening of the chest for therapeutic measures It is little wonder that no progress was made in the therapy of intrathoracic visceral disease until this fact was appreciated in the monumental work of Sauerbruch, around 1900 He ap-

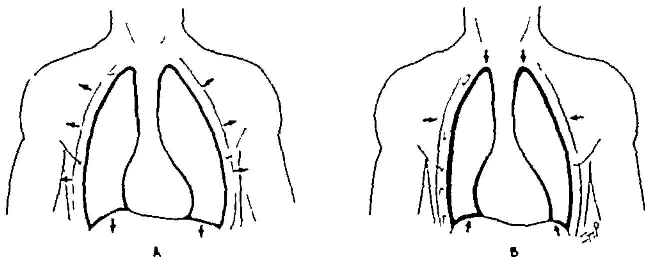


FIG. 24 Normal ventilatory dynamics. *A* Inspiration the chest is enlarged by muscular action upon the rib cage and by descent of the diaphragm. By compliance the lungs are enlarged as they follow the cage outward thus drawing air into the tracheobronchial tree *B* Expiration at the end of inspiration the chest cage recoils and the diaphragm rises. Because the lungs are resilient, air is expelled from the tracheobronchial tree

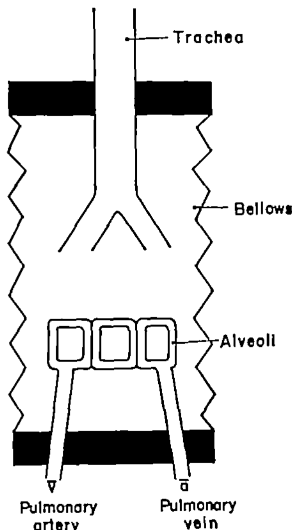


FIG. 25 Schema of the respiratory system The chest wall its musculature, and the diaphragm are represented as a bellows. v mixed venous blood a, mixed arterial blood. (*Courtesy Gordon Snider from Zimmerman and Levine Physiologic Principles of Surgery Philadelphia W B Saunders Co 1957*)

preciated the necessity for maintaining the negative pressure which normally is present in the pleural space For this purpose he operated through a chamber which was maintained at a constant negative pressure by suction pumps Of course this is much more easily accomplished today by the use of *positive pressure anesthesia* with an obliterating cuff over the endotracheal tube. Under such conditions the positive pressure applied by the anesthetist and his machine results in inflation of the lungs by positive pressure and the same result is obtained as if the lungs were being drawn out by the chest wall through an increased negative pressure of the pleural space Only because of this advancement is it possible to operate freely within the thorax today By the same token it must be appreciated that in any wound of the thorax the normal pressure relationships must be restored as soon as possible in order to restore normal ventilatory and respiratory function.

TRAUMA TO THE THORAX

Types—Blunt, Penetrating, and Perforating

Injuries to the thorax and its contained viscera may be sustained in several ways *Blunt force* usually is productive of fractured ribs A single rib or several may be broken Derangement in the physiology of respiration may vary from slight to maximal. There may be no injury to intrathoracic viscera, or injury to heart, lungs and blood vessels may be very severe In civilian life blunt trauma to the chest is commonly found in automobile and industrial accidents *Penetrating injuries* of the thoracic cavity are usually due to sharp or pointed instruments, usually introduced into and withdrawn from the thoracic cavity Such weapons lacerate tissues *Perforating injuries of the thoracic cavity are usually due* to high velocity missiles such as bullets. A wound of entrance and a wound of exit are usually found on initial examination A knowledge of the contents of the thorax and their relation to the surface topography is necessary in assessing possible visceral injuries

Clinical Findings—Diagnosis, Pathology, and Treatment

Fracture of Ribs The most common thoracic injury is the fracture of a single rib Any type of blunt trauma may be responsible Diagnosis is usually made on the history of such trauma and the symptoms of pain in the area of the fracture which is aggravated by motion and deep breathing There usually is local tenderness, and crepitus may be elicited Compression of the rib

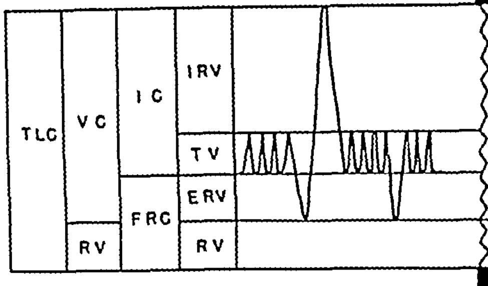


FIG 26 Normal lung volumes and capacities. The volumes do not overlap and The capacities are made up of two or more volumes (IRV) inspiratory reserve v tidal volume (ERV) expiratory reserve volume (RV) residual volume (TLC) pacity (VC) vital capacity (IC) inspiratory capacity (FRC) functional resid Note that in the normal spirographic tracing the vital capacity is an almost ve line (*Courtesy Gordon Snider from Zimmerman and Levine Physiologic Prin gery Philadelphia W B Saunders Co.,* 1957)

cage increases the pain There may be little or no separation at the site of fracture, the parietal pleura may not even have been torn On the other hand, the broken end of the rib may have penetrated the visceral pleurae and lacerated t may be true even though an x tion of the thoracic cage may

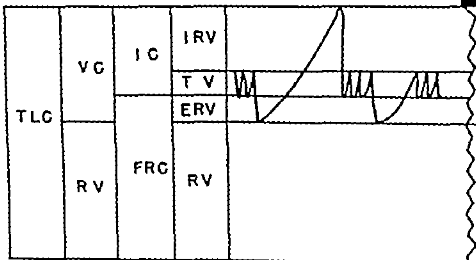


FIG 27 The lung volumes and capacities in obstructive ventilatory dysfunction. volume (RV) and functional residual capacity (FRC) are greatly increased, alth lung capacity is normal The vital capacity (VC) and expiratory reserve volume decreased. Note the marked delay in the performance time of the vital capacit

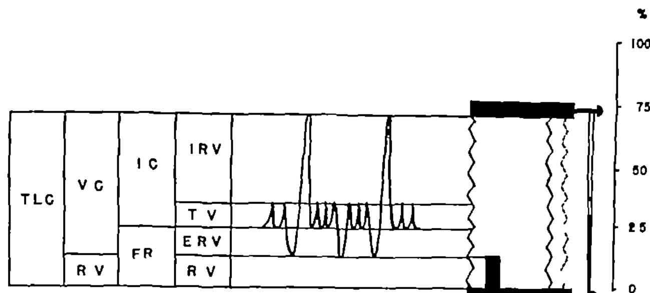


FIG. 28 The lung volumes and capacities in restrictive ventilatory dysfunction The total lung capacity (TLC) is decreased. The bellows is depicted as being reduced in volume and restricted in distensibility The functional residual capacity (FRC) residual volume (RV) and vital capacity (VC) are reduced. There is no delay in performance of the vital capacity (*Courtesy Gordon Snider from Zimmerman and Levine Physiologic Principles of Surgery Philadelphia W B Saunders Co* 1957)

displacement, the penetrating rib fragment having recoiled to almost its normal position Thus the pathologic derangement may be minimal or may include all the complications of punctured lung Even without laceration of the lung the "wet lung" syndrome may occur This refers to the occurrence of pulmonary edema, even on the contralateral side and may occur with even the most innocuous looking rib fractures Treatment of simple rib fractures is directed toward the restoration of normal respiratory physiol

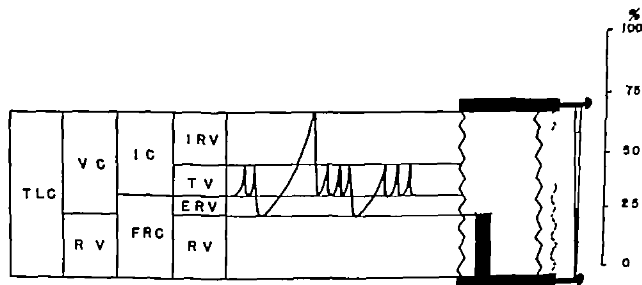


FIG. 29 The lung volumes and capacities in *combined* restrictive and obstructive ventilatory dysfunction The total lung capacity (TLC) is decreased. The residual volume (RV) and functional residual capacity (FRC) are large both in relation to the total lung capacity (TLC) and in absolute terms. There is marked delay in the performance of the vital capacity (*Courtesy Gordon Snider from Zimmerman and Levine Physiologic Principles of Surgery Philadelphia W B Saunders Co* 1957)

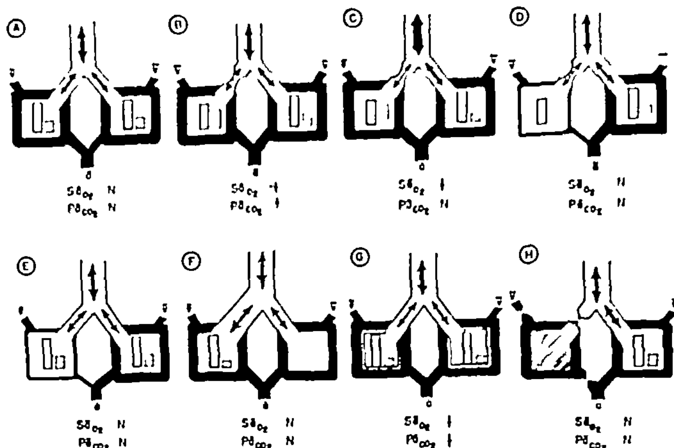


FIG. 30 Schema of normal and abnormal states of alveoli The following symbols are used the stippled area in the bronchioles represents the respiratory dead space the thickness of the vertical arrow in the bronchiole represents total ventilation the thickness of the two smaller arrows represents alveolar ventilation the solid black represents the alveolar capillary with mixed venous blood (v) entering at the top and mixed arterial blood (a) leaving at the bottom the solid rectangle represents alveolar partial pressure of oxygen and the dotted rectangle alveolar partial pressure of carbon dioxide $SaCO_2$ represents saturation of arterial blood with oxygen, *Pa*CO partial pressure of carbon dioxide in arterial blood. *N* = normal —↓ = decreased —↑ = increased. *A* Schema of two normal alveoli with their bronchioles. *B* Regional hyperventilation due to airway obstruction Regional bronchiolar obstruction is depicted by narrowing of the air passage to one alveolus. This results in reduced alveolar ventilation, with an increase in alveolar PCO_2 and decrease in alveolar PO_2. There is a consequent reduction in *Sa*O. The increase in *Pa*CO may cause hyperventilation which is shown in *C* *C* Regional bronchiolar obstruction with compensatory hyperventilation The hyperventilation decreases PCO_2 and increases PO_2 in the normal alveoli with resultant elimination of excess CO from arterial blood. Hyperventilation cannot compensate for the decreased SaO_2 of the blood coming from the hypoventilated alveoli and SaO_2 remains low *D* Reduced capillary perfusion with regional hypoventilation. There is a decrease in blood flow through the capillary of the hypoventilated alveolus, and as a result the regional ventilation has no effect on mixed arterial blood. *E* Reduced capillary perfusion with normal ventilation Alveolar ventilation is normal but there is a regional decrease in capillary blood flow The effect is to increase the respiratory dead space without effect on arterial blood gases. *F* Regional hyperventilation with normal capillary perfusion. Capillary blood flow is normal but there is regional hyperventilation of alveoli which results in increased respiratory dead space *G* Decreased maximum diffusing capacity for oxygen owing to alveolar capillary block This results in a marked depression of SaO_2. There is also hyperventilation which causes a fall in alveolar and arterial PCO_2. The latter is possible because the diffusibility of CO_2 is so much greater than that of oxygen. *H* Decreased maximum diffusing capacity of the lung is due to loss of area of functioning alveolus in contact with functioning capillary (*Courtesy Gordon Snider from Zimmerman and Levine Physiologic Principles of Surgery Philadelphia W B Saunders Co* 1957)

ogy This is best accomplished, not by strapping but by the relief of pain by local anesthetic injection around the appropriate intercostal nerve The control of pain by this technic permits normal ventilatory excursion of the affected hemithorax and prevents noxious reflexes from causing pulmonary edema.

Flail Chest. Crushing automobile and industrial accidents have made the occurrence of *flail chest* a common finding in emergency rooms throughout the country Flail chest exists when so many ribs on both sides have been broken that the function of respiration is grossly disturbed the chest is unable to expand when it should it is "flail," or "stove in. *Paradoxic respiration* is the outstanding finding under these conditions During the inspiratory effort the thoracic cage actually collapses rather than expands This type of respiration may be seen on both sides, on just one side or in just one portion of one hemithorax. Emergent care is necessary to restore ventilatory function and prevent rapid asphyxia. This is accomplished by traction on the ribs and sternum by any available means so that the cage wall cannot fall inward during inspiration. Towel clips pins, or other appliances used to grasp flail portions of ribs and sternum are attached to traction apparatus until fixation has occurred Of greatest importance until such fixation is attainable is the use of a positive negative pressure machine to inhale or exhale for the patient while he cannot do so himself It may be lifesaving Such machines are most advantageously used when combined with tracheostomy The tracheostomy is of further value for the mechanical removal of secretions from the tracheobronchial tree which the patient is unable to bring up himself because of pain and loss of tussive power The removal of such secretions is imperative to prevent atelectasis and collapse

Hemothorax. Hemothorax is the term applied to the presence of blood in the pleural space causing separation of the parietal and visceral pleurae and partial compression of the lung It is most commonly due to penetrating or perforating injuries Laceration of the lung, as stated above can also be caused by blunt trauma, if a rib fragment punctures the lung The blood may come either from the lacerated lung or from a parietal vessel such as the branches of the intercostals or internal mammaries Minimal hemothorax may produce no subjective manifestations whatever If massive (over 500 ml), signs of lung compression, especially dyspnea, will usually be present. Shock is rarely seen with unilateral hemothorax without associated injuries, because penetration of a vessel large enough to produce shock is usually fatal before the patient reaches the emergency room *Occasional massive hemothorax which is remediable by open operation* is seen but such conditions constitute probably only 10 per cent of this type of thoracic emergencies. In all situations of hemothorax roentgen-ray study as well as careful physical examination and frequent assessment of the general condition are essential

There is general consensus that the treatment of choice in hemothorax is the removal of the blood from the pleural space by aspiration The expansion of the lung and the quantity of residual intrapleural fluid should be checked frequently both clinically and roentgenographically Significant reaccumulation of blood is again treated by aspiration. Usually such evacuations are not required more often than once in 24 to 48 hr The re-expansion of the lung by the removal of blood is beneficial in two ways First, the expanding lung is better able to seal off the source of bleeding In addition the expanded lung is less subject to atelectasis in the early days following trauma and to compression of the lung by fibrothorax later Blood that is not removed often results in the deposition of its fibrin on the visceral pleura owing to the constant motion of the lung such deposition, even if not infected, may result in the *compressed lung syndrome* or *fibrothorax* and require later operative intervention

Pneumothorax Closed *pneumothorax* is the presence of air in the pleural space sep-



FIG 31 *A Spontaneous pneumothorax in a patient with bullous emphysema. B Same case, 48 hr later The lung has been re-expanded by placing a tube in the pleural space and applying suction to it.*

arating the visceral and parietal pleurae and preventing the lung from complete expansion. A limited pneumothorax is not disabling and often goes unnoticed by the patient the air is absorbed in a few days If the accumulation of air in the pleural space, however is recurrent or is of such proportions as to compress an appreciable part of the lung then dyspnea is a common early symptom. Treatment, as in hemothorax, consists in the aspiration of the air by needle usually with the patient in the upright or semireclining position the needle is introduced in the second intercostal space anteriorly Aspiration may be repeated at intervals of hours to days. The desired end result is the complete *re-expansion of the lung* with each respiratory excursion (Fig 31)

When both blood and air have escaped from the lung, the findings include an air fluid level on roentgen ray examination. The treatment of this *hemopneumothorax* is the same as if each component had occurred by itself

Tension Pneumothorax. When, owing to either disease (tuberculosis compensatory emphysema, bullous emphysema, pneumatocele pulmonary cyst) or trauma, air escapes from the lung or the bronchi into the pleural space and cannot find egress back *into the tracheobronchial tree* or to the outside the pressure of that air *within the pleural space* progressively rises. This is known as *tension pneumothorax* The mechanism resembles the action of a *flutter valve* at the site of perforation, in that air escapes from the lung and a flap of tissue prevents its returning through the same hole

The signs and symptoms of tension pneumothorax may become rapidly alarming, with dyspnea which may progress to asphyxia of the patient, while at the same time serious cardiovascular disturbances take place The latter are due to the pressure of intrapleural air displacing the mediastinum and its contained structures, the heart and great blood vessels, to the opposite side, with compression of the great veins entering the heart. This inflow stasis eventuates in tachycardia and diminished cardiac output.

The treatment of tension pneumothorax is emergent in nature. A needle introduced into the pleural cavity through the second

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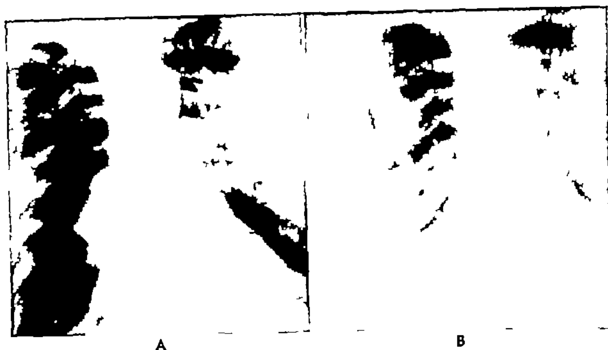


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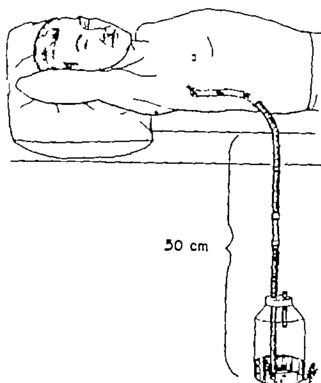


FIG. 32. Water-sealed drainage of the pleural space. Expelled fluid is forced into a bottle by expansion of the lung Expelled air bubbles out the open short glass tube For complete safety the bottle is placed about 50 cm below the level of the chest. If it were only 10 to 15 cm below the chest water in the bottle would be aspirated in the pleural space.

intercostal space anteriorly will relieve the pressure If the expanding lung then prevents further escape of air nothing further is needed However if the tension pneumothorax recurs the pleural space must continuously be decompressed by providing means of egress of air A catheter is inserted and connected to a water-sealed bottle placed on the floor in which the pleural air is trapped (Fig 32) Since normal inspiratory pressure is about 15 cm H_2O the bottle must be placed more than this distance below the level of the chest it is preferable to have it placed 40 to 50 cm below the level of the chest If the air escapes rapidly from the lacerated or diseased lung, it may be necessary to apply suction to the water sealed trap in order to remove the air from the pleural space more rapidly than it enters If such an air trap mechanism does not resolve the pneumothorax open operative intervention to close the site of air leak must be carried out.

Pneumomediastinum and Tension Pneumomediastinum Occasionally the rupture of a large bronchus will cause the discharge of air into the mediastinum rather than into the pleural space Such a *pneumomediastinum* may occur following rupture of emphysematous blebs as well without the production of pneumothorax The anatomic basis of such occurrence lies in the fact that rupture of blebs within the depth of the lung may cause the dissection of air along the perivascular sheaths of the pulmonary vessels into the mediastinum without the air breaching the visceral pleura. If the leak is transitory and slow enough that the air may be absorbed, no permanent harm may result. If however the air accumulates within the mediastinum at a rate faster than the loose areolar tissue of that compartment can absorb it, a state of tension will exist, i e., *tension pneumomediastinum* This constitutes one of the most dire emergencies in thoracic surgery because compression of the great vessels within the mediastinum particularly of the veins rapidly reduces cardiac inflow An alarming clinical picture results asphyxia, severe dyspnea, cyanosis, distended neck veins and cerebral anoxia. Emergency mediastinotomy the opening of the anterior superior chamber of the mediastinum by a small incision in the suprasternal notch with finger dissection of the deep fascia, will release the air with an audible hiss and with a spectacular recovery The diagnosis of tension pneumomediastinum must be made soon after it occurs and treatment carried out immediately if it is to be lifesaving. Total traumatic rupture of the trachea or a main bronchus, as a result of automobile accidents, is occasionally seen to produce this serious picture

Sucking Wounds. When a hole is made in the thoracic cage the mechanism of ventilation of the lung on that side is destroyed Each attempt at inspiration draws air into the aperture in the thoracic wall instead of into the lung through the orotracheobron

Serial electrocardiography reveals changes suggestive of myocardial damage which are of diagnostic import These lesions are frequently unrecognized until the patient, who is apparently recovering from associated injuries dies unexpectedly The increasingly frequent demonstration of cardiac trauma at postmortem emphasizes the necessity of awareness of this possibility by those giving initial care in the emergency room

Combined Thoracoabdominal Injuries. Severe violence to the trunk frequently results in simultaneous injuries to viscera above and below the diaphragm Those in the abdominal cavity more often require operative intervention because of the possibility of rupture of a hollow viscus with its attendant peritonitis or the continued bleeding from a solid viscus such as the spleen and liver In stab or gunshot wounds the path of the weapon or the *trajectory* of the missile must be studied very carefully to determine which viscera may have been *injured* *Even when both the wounds of* entrance and exit are on the thoracic cage all serious injury may be below the diaphragm involving such structures as the liver spleen, stomach and colon The changes in topographic relationships with the excursions of the diaphragm must be considered

The diaphragm itself may be breached, thereby converting the pleural and peritoneal spaces into a single cavity Blunt violence with severe crushing of the chest or abdomen may rupture the muscle and force abdominal contents into the chest with serious embarrassment of cardiac respiratory and gastrointestinal functions. Penetration of the diaphragm may not be immediately recognized and may give rise to symptoms of diaphragmatic hernia at a later period In the management of thoracoabdominal trauma operative exposure of one cavity may disclose a wound of the diaphragm which requires inspection and treatment of viscera in the other compartment This may frequently be achieved through the diaphragm Whenever the pleural space is opened, by whatever approach including transdiaphragmatically, it must be drained by a water sealed method.

PLEURAL EFFUSION

Types

Serous and Sanguinous. Pleural effusions the accumulation of fluid in the pleural space separating the visceral from the parietal pleurae are of several types. As seen above the effusion may be extravasated *blood* it may also be *serous* in nature or the serum-colored effusion may have some blood in it, constituting a *serosanguinous* effusion

Purulent. When fluid in the pleural space becomes *purulent* the effusion takes the name *empyema* In most instances empyema represents a suppurative infection of a serous effusion, which usually could have been obviated by adequate therapy There is a gradual transition, with increasing quantities of fibrin inflammatory cells, and debris *until the fluid appears as pus*

Chylous Chylous effusion results when the lymph flowing in the thoracic duct on its way to junction with the venous system, is spilled into the pleural space Any accumulation of fluid in the pleural space may be either *free* or *encapsulated* *In the former* case it is dependent, resting on the diaphragm when the patient is in the upright position and posteriorly as he lies supine in bed An accumulation of fluid may be encapsulated at any place within the pleural space depending on the deposition of fibrin and the formation of synechiae between the visceral and parietal pleurae A common place for encapsulation is in the interlobar space

Clinical Causes

Trauma and Cardiac Decompensation The pleural effusion consequent upon

trauma is almost always bloody as was described in the section above Accidental or surgical trauma may also be followed by chylous effusion due to tearing of the thoracic duct Of all pleural effusions the overwhelming majority are consequent upon *cardiac decompensation* and the fluid is a serous *transudate* The diagnosis is usually simple because of the associated circulatory findings and the physical evidence of the pleural effusion

Secondary to Lung Pathology Pulmonary disease constitutes the second large group of diseases which give rise to effusion in the chest. Both medical and surgical therapy may be required *Pneumonia* was a common cause of pleural effusion before the days of chemotherapy and biotherapeutic agents and it is not uncommon today Since this disease is actively inflammatory the pleural effusion is an *exudate* and may be serous, fibrinous, or purulent, depending upon the stage and the effectiveness of therapy Staphylococcus pneumonia is a frequent cause of pleural effusion particularly in children *Pulmonary tuberculosis* while less prevalent than even a decade ago still is responsible for many pleural effusions Aspiration and study of pleural fluid for acid-fast organisms come early in the differential diagnosis of an effusion of unknown origin Suppurative pleural exudate may be consequent upon *pulmonary abscess* and is not infrequently seen in *chronic bronchiectasis*

Secondary to Lymphatic Obstruction and Diaphragmatic and Subdiaphragmatic Pathology Pleural effusion is frequently seen in primary and secondary *pulmonary carcinoma* especially when the lesions are subpleural and most particularly when the entire pleura is seeded with carcinoma (carcinomatous pleurae) The effusion may be due in part to obstruction of the lymphatics in the pleura which is a single layer of mesothelium Accumulation of fluid in the pleural space is occasionally due to disease below the diaphragm. The common basis is *subdiaphragmatic abscess* An unusual type of serous effusion occasionally is seen in the thorax associated with a fibroma of the ovary (Meigs syndrome), however, this type of effusion has been seen with other intraperitoneal tumors as well The mechanism is not well understood.

Treatment of Effusions

The therapy of pleural effusion is directed along two main lines (1) the treatment of the underlying disease to which the effusion is secondary (2) aspiration of the fluid for two purposes (*a*) for identification by cytologic examination, culture and chemical study and (*b*) for the relief of dyspnea due to compression of the lung Empyema presents problems of a distinctive nature and will be discussed in the following section.

Course and Sequelae

Pleural Thickening Small pleural effusions are absorbed particularly if the fluid is a transudate this obtains most commonly when the cause is cardiac decompensation and the therapy is effective This may be true also when the fluid is an exudate and the inflammatory disease to which it is secondary is overcome by appropriate treatment. Large accumulations of fluid, however if not removed may result in one of three sequelae, with varying degrees of subsequent disability These are in order of increasing severity (1) pleural thickening, (2) empyema, and (3) fibrothorax. There may be only a residual parietal *pleural thickening* which may be somewhat confusing in the interpretation of future x ray pictures of the chest but usually is not very disturbing to pulmonary function

Empyema If a pleural effusion is infectious in nature that is, contains viable microorganisms, and is not properly treated by aspiration and the systemic use of biotherapeutic agents, it may be converted into pus, which, in a mesothelium-lined cavity is called *empyema* In addition to the signs

and symptoms of a pleural effusion there is now the finding of pus on aspiration plus the systemic manifestations of an infectious process fever, leucocytosis, and toxemia

Treatment of Empyema and Fibrothorax

Closed The treatment of empyema has undergone great changes during the past two decades During World War I and in the years intervening until World War II empyema was treated by means which are considered less than satisfactory today Aspiration by needle was done then as now both for the identification of bacteria in the material and to remove as much of it as possible Usually the effusion became thick and fibrinous very rapidly permitting no further evacuation by needle The next step consisted in placing a catheter through an intercostal space through which a liquefying solution was sometimes used to lyse the fibrinous adhesions and make the pus more fluid so that it might be aspirated through the tube The morbidity of empyema remained high Even open drainage which involved the resection of a small fragment of rib was inadequately accomplished. The unwillingness of the surgeons of that day to do an open thoracotomy in the presence of pus in the pleural space is easily understood, since without coverage of chemotherapeutic and biotherapeutic drugs the mortality rate was appreciable and the morbidity of such a procedure was very high

Enzymatic Debridement and Open Operation Modern therapy of empyema begins in a fashion similar to that of the previous era but rapidly becomes more extensive Aspiration is done very frequently as often as daily in order to remove as much of the infectious effusion as possible, to identify it and to instill appropriate antimicrobial drugs into the pleural space through the same needle If this is unsuccessful and the diagnosis of an empyema with thick pus is made or if the patient comes to one's care at a time when the diagnosis of fibrothorax can be made the operation for the cure of this disease is much more extensive and much more curative As a preoperative measure *enzymatic debridement* may be employed This involves the intrapleural injection of materials classified as *fibrinolysins* which are occasionally successful in thinning the purulent material and the breaking down of loculations. Materials most commonly used are derivatives of the streptococci (streptokinase streptodornase) and Tryptar a proteolytic enzyme Occasionally early in the process of the development of an empyema, the use of these materials may avoid operation If operation is required, it involves open thoracotomy under the protection of appropriate antibiotic treatment, the evacuation of pus in the pleural space and the operation of decortication in addition With good preoperative management, blood and blood substitutes, accurate fluid and electrolyte balance and proper positive pressure endotracheal anesthesia, the results of treatment of empyema and fibrothorax by open operation and decortication are highly successful

Fibrothorax and Decortication. If the empyema is not adequately evacuated and the infection successfully combatted, a pyogenic membrane forms which subsequently becomes a fibrous encasement over the visceral pleura which will compress the lung This complication has earned the name of *fibrothorax* indicating the fibrous sheath which encompasses the visceral pleura and the lung. It gives rise to the symptoms of *lung compression* which are pulmonary insufficiency in general manifested by dyspnea, tachycardia, and respiratory lag on the affected side The therapy of a well-established fibrothorax is exclusively surgical requiring the operation of *decortication* In this procedure the fibrous sheath is removed from the visceral pleura thus allowing the lung to expand normally

The dividing line in time between acute and chronic in many diseases is difficult to define However in the case of fibrothorax it is somewhat more precise in that anywhere up to 6 weeks after the establishment

of the empyema the fibrothorax is relatively easy to remove from the visceral pleura coming off like the peel of an orange After the sixth week however it is extremely difficult to decorticate such a lung without tearing the visceral pleura and opening small air leaks Accordingly decortication is best accomplished before it becomes chronic i e within the first 6 weeks after the em pyema becomes established

SUMMARY OF PRINCIPLES

1 Logical and successful therapy of wounds and diseases of the chest are dependent upon the knowledge of the physiology of normal respiration (ventilatory phase and respiratory phase) and in understanding of the pathologic physiology involved

2 Trauma to the thorax may occur as a result of blunt force or penetrating and perforating weapons

3 A single fractured rib is the most common thoracic injury It may be associated with laceration of the lung and complications within the pleural space, or it may not even be detectable by x ray examination The sequelae of fractured ribs may include limitation of respiratory excursion owing to pain which may be followed by atelectasis the "wet lung" syn drome which is pulmonary edema may occur even on the contralateral side

4 A flail chest is one in which so many ribs have been broken that the thoracic cage does not expand on inspiration instead, paradoxic respiration the falling in of the chest, occurs with each inspiratory move ment Early stabilization of the thoracic cage by external fixation is life saving and a positive negative respiration machine is most helpful

5 Hemothorax is the accumulation of blood in the pleural space The blood should be removed in order to allow the lung to expand and func tion normally and to prevent the occurrence of fibrothorax

6 Pneumothorax is the accumulation of air in the pleural space. It may be due to air introduced from without or the escape of air from a lacerated or diseased lung A small pneumothorax need not be removed A large pneumothorax must be removed to prevent compression of the lung

7 Tension pneumothorax is the presence of air in the pleural space under increasing positive pressure It is an emergency in that it (*a*) com presses the lung and (*b*) produces movement of the mediastinum away from the side of the pneumothorax with potential compression of important structures in the mediastinal compartment and the opposite hemithorax The effective treatment of tension pneumothorax is the underwater sealed drainage of the air from that pleural space and the closure of the defect from which the air is continuously escaping

8 Air may escape from the tracheobronchial tree and from the alveoli into the mediastinum instead of into the pleural space Such an accumu lation may be under tension (tension pneumomediastinum) a dire surgical emergency treated by anterior superior mediastinotomy and treatment of the underlying disease or injury

9 Sucking wound of the chest must be closed as an emergency even before patients are transported to the hospital in order to prevent pulmonary insufficiency and the deleterious effects of mediastinal flutter *Trauma by compression to the superior mediastinum anteriorly may cause* diffuse hemorrhage in the brain, head, and neck, with an area of cyanosis above the clavicles

10 Damage to the heart from myocardial contusion is a potential serious injury whenever severe blunt trauma is sustained over the sternum

11 Injury to viscera both above and below the diaphragm occurs in blunt trauma to the trunk and in penetrating and perforating injuries Operative intervention is necessary more frequently for injuries to abdominal viscera than to those of the thorax.

12 Effusions into the pleural space may be serous, sanguinous, purulent, or chylous. The accumulations of fluid may be dependent, free or encapsulated anywhere within the pleural space. Hemothorax must also be included among the effusions

13 Clinical causes of pleural effusion include trauma open operation, diseases of the lungs themselves (pneumonia tuberculosis, carcinoma, abscess) cardiac decompensation, and subdiaphragmatic disease

14 The treatment of pleural effusions is the treatment of the underlying disease plus the removal of the fluid for identification culture cytologic examination and relief of lung compression

15 The sequelae of pleural effusion are pleural thickening, empyema, and lung compression due to fibrothorax

16 The early treatment of empyema is the removal of the fluid and the instillation of antibiotics into the pleural space. Enzymatic debridement may help in the process of liquefying fibrinous adhesions and synechiae If the empyema cannot be drained by this means open operation is performed under cover of antibiotic therapy the purulent material is removed and decortication of the lung is accomplished.

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Tumors of the Lungs and Bronchi

I Carcinoma of the lung
  A Etiology (1) carcinogens (2) metaplasia
  B Pathology (1) histology (a) squamous (b) adenocarcinoma (c) oat cell (d) undifferentiated (2) progress of disease (a) extension (b) lymphatic spread (c) hematogenous spread (d) aerogenous spread (3) intrapulmonary pathogenesis (a) atelectasis (b) pneumonitis
  C Diagnosis (1) history (2) symptoms (3) signs (4) x ray examination (5) exfoliative cytology (6) scalene node biopsy (7) bronchoscopy with biopsy (8) exploration
  D Differential diagnosis (1) tuberculosis (2) other granulomas (3) pulmonary cysts (4) emphysematous bullae (5) gastroenteric cysts (6) aneurysms (7) metastatic tumors
  E Treatment
  F Prognosis
II. Metastatic tumors: bone, thyroid, kidney breast, and prostate

CARCINOMA OF THE LUNG

Etiology

The spotlight of medical attention is sharply focused on cancer of the lung This dread disease considered little more than a medical curiosity only a generation ago now looms as the most common cancer in the adult American male As elsewhere the true cause of carcinoma is not known. However two factors can be implicated beyond any reasonable doubt. The first is the influence of certain inhalant *carcinogens* including cigarette smoke and many industrial irritants, which can be shown to be capable of irritating bronchial epithelium toward tumor formation The role of smoking specifically cigarette smoking in the rapid increase of pulmonary cancer has been widely discussed in the lay press as well as in medical literature The cumulative evidence is overwhelming Continued studies of this relationship should point the way toward means of reducing the frightful suffering and fearful mortality of cancer of the lung The *second prominent factor* as a precipitating agent is the *metaplasia* of bronchial epithelium which has undergone repeated *suppurative disease* and healing such as is found in chronic bronchiectasis The increased frequency of pulmonary carcinoma is real and *not merely* apparent because of better means of detection.

Pathology

Almost all tumors of the lung are of epithelial origin. An occasional malignant mesenchymal tumor is seen, but they are rare The carcinomas of the lung probably *all arise in the* epithelium of the bronchial mucosa. Some pathologists doubt that alveolar cells are ever the site of origin of any

of these malignancies. The terms *carcinoma of the lung* and *bronchogenic carcinoma* are therefore, used in the literature interchangeably

The histologic types of bronchogenic carcinoma, in ascending order of degree of malignancy are (1) squamous cell, (2) glandular (adenocarcinoma) (3) oat cell and (4) undifferentiated. *Squamous* or *epidermoid* is the most common of all types of lung cancer and is the specific form which has so increased in frequency in men who have smoked heavily over a period of many years. *Adenocarcinoma* appears to be not related to heavy smoking and is much less common It is practically the only type found in females The *oat cell* variety is a highly cellular tumor which histologically resembles sarcoma and frequently infiltrates the lung diffusely The *undifferentiated cell carcinoma* as might be expected is the most malignant of all known types Both of the last two named varieties are highly malignant and rapidly invasive and cures by any method are rare

The disease progresses in any of several methods. *Extension* may occur to adjacent structures including the hilar lymph nodes, esophagus, large vessels of the mediastinum, parietal pleura, and the chest wall. *Lymphatic spread* may also carry the tumor via the mediastinum into the lymph node chain in the neck. Similarly lymphatic spread via the diaphragm and falciform ligament may occur into the liver *Hematogenous dissemination* occurs most commonly to the kidney liver brain and skeleton. *Aerogenous spread* can occur from bronchus to bronchus within one lung or across the carina to the contralateral lung

The initial lesion leads early to obstruction of smaller bronchioles with the production of atelectasis in the alveoli which they serve Pneumonitis is frequent in the atelectatic lung

Diagnosis

The diagnosis of carcinoma of the lung is made on the sequence of the following typical history symptoms, signs, and laboratory findings Unfortunately symptoms may be absent or too mild to impress themselves on the attention of the patient, and the lesion may be detected accidentally during routine roentgenography of the chest. The cardinal symptoms, which are often late manifestations, are chronic cough, chest pain hemoptysis, and dyspnea Of these the most common is *cough* which is usually dry, but which may be productive of blood. It is occasioned by endobronchial irritation by the growing mucosal lesion and the consequent atelectasis. The *pain pleurodynia* is due to involvement of the visceral pleura The patient may have noticed *wheezing respiration* as the only symptom This is explicable by expiratory bronchiolar obstruction *Hoarseness* is a late sign and usually indicates involvement of a recurrent laryngeal nerve *Dysphagia* is occasionally the presenting symptom and is due to extrinsic compression of the esophagus by extension of the tumor

The signs that are elicited in the examination of the patient are referable to the progression of the disease within and beyond the confines of the lung *Dyspnea* may be due to either pleural effusion with lung compression or obstruction of a relatively large bronchus or of many small bronchioles. Dullness and suppression of breath sounds may lead to discovery of the atelectatic area. A supraclavicular mass may be a metastatic node, particularly common in superior sulcus tumors (Pancoast) Accompanying this tumor Horner's syndrome is also frequently found it is due to involvement of the cervical sympathetic chain

X ray examination may reveal an area of atelectasis of varying extent which in itself is not specific. It may also disclose an effusion which then must be subjected to further diagnostic procedures as indicated *in a previous chapter* The roentgenogram may reveal a space-occupying lesion which may be discrete or diffuse and fan-shaped It may be situated centrally or peripherally *Exfoliative cytologic examination* the ex

amination of the sputum for desquamated cells, has become one of the most useful laboratory procedures for the diagnosis of this tumor

Biopsy of a palpable scalene node may be of even more value than exfoliative cytology In a fair percentage of examinations, biopsies of the scalene fat pad disclose lymph nodes which are not palpable and which occasionally reveal invasion by carcinoma of the lung Some clinics report the use of scalene fat pad biopsy as a routine others seek a biopsy at this site only if a scalene node is palpable

Endoscopic examination of the tracheobronchial tree (bronchoscopy) with biopsy of suspicious appearing tissue and always with aspiration of material for examination by the Papanicolaou technic is always done if diagnosis is not made by other methods.

When all the above methods have failed to make a diagnosis of a space-occupying lesion, it is considered obligatory to perform an exploratory thoracotomy With present technics this operation is no more hazardous than is exploratory celiotomy

Differential Diagnosis

The differential diagnosis of carcinoma of the lung includes all space-occupying lesions within the thorax, as well as all conditions producing pulmonary symptoms among them (1) bronchial adenomas, which usually give only endobronchial symptoms and are actually or potentially carcinomas (2) tuberculosis (3) other granulomas of the lung including fungus diseases (4) bronchiectasis and abscess (5) pulmonary cysts usually congenital (6) emphysematous bullae (degenerative disease) (7) gastroenteric cysts (embryonic remnants) (8) aneurysms (9) pericardial fat pads (10) occasionally even hydatid cysts and diaphragmatic hernias

Treatment

The treatment of carcinoma of the lung is directed toward cure by extirpation of the involved pulmonary tissue, if this is anatomically feasible and not otherw traindicated by contralateral pu insufficiency or the presence of dist stases. One half of all cases are al operable at the time of initial exa Of the remaining 50 per cent upc thoracotomy is done only half a nically resectable and of these s obviously beyond anything but pall

Prognosis

The prognosis of cancer of the extremely poor The 5-year surviv recurrence) rate is probably not m 10 per cent of all the cases origin i e., 20 per cent of all the cases upon The worst prognoses are in of oat cell and undifferentiated typ clinics will refuse even thoracoto patient in whom exfoliative cytolog doscopic biopsy has disclosed the logic types. The prognosis is pa poor in patients under the age of fo

METASTATIC TUMORS BONE THYROID, KIDNEY, BREAST, AND PROSTATE

Metastatic malignant tumors of may be found on routine roentgen amination as the first intimation th tant primary tumor may exist. On hand, the examination of the lung done in search of metastases when lesions are known to be present el The x ray shadow of metastatic usually more sharply delineated t mary growths they are occasionall but often multiple Metastatic tumo lung are brought there by the blood Certain epithelial neoplasms have a predilection for pulmonary me These include lesions of bone, thyro kidney (clear-cell carcinoma hy roma) breast, and prostate N growths of the gastrointestinal tract uncommonly seen to metastasize lung Sarcomas which also characte spread by the hematogenous ro found frequently to metastasize to tl

SUMMARY OF PRINCIPLES

1 Cancer of the lung is now the most common cancer of the adult male Known carcinogenic factors include cigarette smoke and industrial irritants the cumulative evidence for this fact is overwhelming Metaplasia of bronchial epithelium in chronic pulmonary suppurative disease may also be contributory

2 The terms *carcinoma of the lung* and *bronchogenic carcinoma* are *used interchangeably since most if not all lung tumors* arise from bronchial epithelium Mesenchymal tumors are rare

3 Squamous or epidermoid bronchogenic carcinoma is the most common of all types of lung cancer and is the specific form which has so increased in frequency in men who have smoked heavily over a period of many years

4 Adenocarcinoma of the lung appears to be not related to heavy smoking is much less common and is practically the only type found in females.

5 The *oat cell (or small cell) variety of bronchogenic carcinoma* is a highly cellular tumor which histologically resembles sarcoma and which infiltrates the lung diffusely The undifferentiated cell carcinoma is the most malignant of all types In these two types cures by any method are rare

6 Bronchogenic carcinoma progresses by extension to adjacent structures in the mediastinum by lymphatic spread along drainage areas into the neck and to the diaphragm by hematogenous dissemination to kidney liver brain, and skeleton and via the aerogenous route from bronchus to bronchus.

7 Carcinoma of the lung must be suspected in every case of cough, pain and hemoptysis The initial symptoms are occasioned by endobronchial irritation by the growing mucosal lesion and by the atelectasis it produces Dysphagia, hoarseness and dyspnea are late signs and indicate extensive disease

8 Bronchoscopy and biopsy of any suspicious lesion exfoliative cytologic examination of sputum and bronchial washings and occasionally exploratory thoracotomy are all used in an attempt to make a diagnosis of cancer of the lung when the lesion may still be amenable to surgical therapy

9 Metastatic malignant tumors of the lung are usually found on routine roentgen ray examination are frequently single but may be multiple and are brought to the lung by the hematogenous route

10 Tumors which frequently metastasize to the lung include lesions of bone thyroid kidney breast and prostate sarcomas metastasize to the lungs by the hematogenous route as a rule

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Pulmonary Infections

- I. **Bronchiectasis**
  - *A* Definition
  - *B* Etiology
  - *C* Pathology
  - *D* Diagnosis (1) symptoms (2) signs (3) laboratory data
  - *E* Treatment (1) prophylactic (2) definitive
  - *F* Complications (1) pulmonary hemorrhage (2) lung abscess (3) bronchopleural fistula (4) brain abscess
- II **Pulmonary tuberculosis**
  - *A* Natural history of the disease
  - *B* Treatment
- III **Nontuberculous lung abscess (pyogenic) and pulmonary gangrene (putrefactive abscess)**
  - *A* Definition
  - *B* Etiology (1) aspiration (*a*) vomitus (*b*) sinusitis (*c*) foreign body (*d*) tonsillectomy (*e*) malignancy (2) bronchiectasis
  - *C* Pathology
  - *D* Diagnosis
  - *E* Differential diagnosis
  - *F* Treatment (1) prophylactic (2) definitive (*a*) acute (*b*) chronic
- IV **Less common pulmonary infections**
  - *A* Fungi
  - *B* Hydatid cysts

BRONCHIECTASIS

Definition

Bronchiectasis the most common pulmonary suppurative disease is characterized by dilatation of the bronchi and superimposed infection with the production of copious quantities of sputum It was originally classified as either congenital or acquired but it is now generally agreed that the type previously thought to be congenital actually starts in infancy and is therefore also acquired

Etiology

Bronchiectasis is a frequent sequel to pneumonia the bronchial wall is infected and weakened by the extreme tussive effort. Purulent material aspirated during sleep particularly in children, from infected para nasal sinuses and giving rise to suppurative bronchitis, ultimately also leads to bronchiectasis. This obtains so often that the term sinobronchial disease is used to express this relationship A retained foreign body particularly of the vegetable or animal variety is not infrequently the causative culprit.

Tuberculosis has sometimes been thought to be etiologically involved in bronchiectasis, even though the characteristic sites of these two diseases are not the same. The tussive effort of the chronic cough of tuberculosis may account for the association with bronchiectasis. Allergic diseases with pulmonary manifestations are also thought to predispose to bronchiectatic disease.

Pathology

Pathologically, the bronchial wall may exhibit either saccular or cylindrical dilatations, and these areas of suppuration are surrounded by zones of fibroid pneumonitis. One or more lobes may be involved, or the disease may be limited to one or more pulmonary segments. The process is more common in the lower lobes and in the lingular division of the left upper lobe. It is thought that these areas are more commonly involved because they drain upward with the body in the erect position. The entire lung or both lungs may be involved (universal bronchiectasis).



FIG 34. Bronchiectasis of the lower lobe of the left lung demonstrated by bronchography.

Diagnosis

The diagnosis is suggested by the presenting symptom of a cough which is productive of much sputum, particularly on arising in the morning. The sputum may be streaked with blood, or there may be frank hemoptysis. The coexistence of an active sinusitis is more commonly present than absent. Examination of the patient reveals the moist cough, productive of a copious sputum which has a sweetish odor and which characteristically separates into several layers when allowed to stand. If the disease process is very extensive or is of long standing, systemic evidence of chronic suppuration is found in the form of a leukocytosis with a relative shift to the left. In chronic pulmonary disease of any type clubbing of the fingers (osteoarthropathy) and polycythemia may be found. There is not infrequently an associated arthritis, as with any long-standing suppurative disease.

Roentgen ray examination of the chest may reveal many areas of cloudiness which are interpreted as either accumulations of purulent material or multiple areas of atelectasis, or both. Bronchoscopy will usually not fail to reveal the infectious changes of the bronchial mucosa and will yield sputum for examination. It also provides an opportunity for making bronchograms. With the injection of radiopaque materials the bronchiectatic cavities can be outlined (Fig 34). Bronchography is particularly important if resectional therapy is under consideration, so that all possible normal lung tissue may be allowed to remain.

Treatment

The therapy of bronchiectasis falls logically into two phases, prophylactic and direct. The prophylaxis of bronchiectasis consists in the vigorous treatment of purulent sinusitis by all appropriate means. The search for foreign bodies, particularly in

children and especially those of vegetable origin and their removal as early as possible are important prophylactic measures The vigorous treatment of pneumonitis and the control of a severe cough may aid in preventing this serious bronchial disease Active therapy of pulmonary manifestations of allergic diseases is advisable

The direct medical management of bronchiectasis includes several modalities. *Postural drainage* should be employed for the emptying of the bronchi of the lower lobes and the lingula. This is particularly advisable in the morning upon arising, following accumulation of purulent material in the lower bronchi during the night. Antibiotics should be employed to control the peribronchial spread of infection Bronchodilators and wetting agents are useful adjuncts in the therapeutic regime they overcome the spasm associated with the infection and reduce the viscosity of the thick secretions respectively They are employed as inhalants. The oral administration of expectorant mixtures is frequently useful the iodides and the guaiacol sulfonates are frequently used for this purpose Definitive surgical therapy of bronchiectasis which does not respond adequately to medical management must frequently be resorted to Resection may include an entire lobe or both lower lobes or may be limted to bronchopulmonary segments of an involved lobe Not infrequently it is necessary to resect portions of both lungs at staged intervals Preoperative evaluation of bronchiectatic disease is one of the most important uses of bronchography

Complications

The complications of inadequately treated bronchiectasis are all of a serious nature *Pulmonary hemorrhage* may be massive or may be less serious and more frequent The most common cause of *hemoptysis* is this disease The extension of a bronchiectatic cavity into pulmonary tissue may result in a *pyogenic pulmonary abscess* Such abscesses may rupture through the pulmonary tissue into the pleural space with the formation of a *bronchopleural fistula* and its resultant *empyema* Death from metastatic *cerebral abscess* is not uncommon

PULMONARY TUBERCULOSIS

Natural History of the Disease

The therapy of human tuberculosis has undergone dramatic change, both medically and surgically in recent years No consideration of the therapy of this widespread chronic human infection can disregard the natural history of the disease Certainly it is true that the microorganism (*Mycobacterium tuberculosis*) is invulnerable to many forms of attack upon it. This is attributable largely to its hard polysaccharide shell which is resistant even to most acids. At the same time it is also true that man has a remarkable ability to contain this disease by natural defensive processes This relative immunity is strongest in populations that have been exposed longest to this invader Human population groups more recently exposed (100 years, as in the case of the immigrant African Negro) have not yet developed such strong resistance and are therefore more violently afflicted with this disease The evolution of surgical procedures in the therapy of pulmonary tuberculosis, culminating in present-day combined utilization of medical and surgical methods, is an interesting chapter in the conquest of one of the world's major medical problems. It further promises that when the chemistry of immunology and the interaction of the proteins of the various living kingdoms are sufficiently understood, surgical therapy will no longer be involved in the treatment of this disease.

Treatment

The essential medical therapy of tuberculosis has always been the provision of rest so that defensive mechanisms of the body may be able to wall off the disease by the process of fibrosis This was accomplished

by placing the entire patient at bed rest followed by graded return to normal activity Surgical therapy of pulmonary tuberculosis attempted to accomplish rest of the lung by mechanical means, diminishing its activity by placing various types of obstruction to its distensibility The earliest and most frequently used procedure was the purposeful production of *pneumothorax* the introduction of air into the pleural space, causing collapse of the lung In disease of the lower lobes (not as common as in the apices) the introduction of air into the peritoneal cavity *pneumoperitoneum* was sometimes helpful, elevated position of the diaphragm prevented excursion of the lower lobes. This was also achieved by *paralysis of the phrenic nerves* by crushing or excision *Scalenotomy* the cutting of the scalene muscles at or near their attachment to the first rib deprived the apex of part of its ability to expand In extensive tuberculosis there are commonly found synechiae between the visceral and parietal pleurae, especially in the region of the apices which prevent the collapse of the apical portions of the upper lobes The procedure of *intrapleural pneumonolysis* the cutting of these adhesions allows the apex to drop down and to be collapsed by an adjunctive method of treatment. More recently this obstruction to apical collapse has been overcome by procedures in which it is not necessary to enter the pleural cavity In *extrapleural pneumonolysis* the parietal pleura and endothoracic fascia are peeled from the rib cage and inert materials placed between the rib cage and the endothoracic fascia (paraffin air-containing plastic spheres) to maintain the desired collapse In the past, when the disease process was so extensive that the collapse of the lung was desired over a long period of time or even permanently the procedure of choice was *thoracoplasty* This operation destroys, in varying degree the ability of the rib cage to perform its ventilatory function Sections of varying number of ribs are removed so that inspiratory expansion of the lung is minimal

Within the last decade under protection of antibiotics particularly effective against *M tuberculosis extirpative surgery* has greatly improved the therapy of tuberculosis, particularly in patients with far advanced unilateral disease Excisional surgery may be limited to a wedge resection of a small area, a segmental resection a lobectomy or pneumonectomy Toward the end of the decade the number of cases subjected to extirpative surgery has again been reduced, with the development of new antimicrobial agents, such as para aminosalicylic acid and isoniazid, particularly in combination with dihydrostreptomycin The use of these drugs has further diminished the morbidity of and complications following the surgery of pulmonary tuberculosis It has also markedly reduced the incidence of bronchopleural fistula and tuberculous empyema. The future development of even better drugs bids fair to narrow the field of pulmonary surgery for this disease even further

The above pleasant outlook into the future in the therapy of advanced pulmonary tuberculosis holds true for the United States and other countries where preventive medicine is of a high order However there remains a tremendous backlog of moderately advanced and far advanced cases of pulmonary tuberculosis in areas that are densely populated and poverty-stricken where many of the procedures mentioned above will still find great usefulness.

NONTUBERCULOUS LUNG ABSCESS (PYOGENIC) AND PULMONARY GANGRENE (PUTREFACTIVE ABSCESS)

Definition

As elsewhere, an *abscess* is an accumulation of pus in a delimited area. Abscesses within the lung are usually either of tuberculous origin or of pyogenic origin. Either may become a putrefactive abscess when gangrene of the lung occurs, i.e. when there is compromise of blood supply

Etiology

The most frequent cause of pulmonary abscess is the *aspiration* into the tracheobronchial tree of material normally foreign to it. A common offender is vomitus, aspirated in the patient unconscious for any reason, most commonly in the immediate postoperative period as anesthetic level rises during coma from any cause (head injuries diabetic coma, alcoholic stupor) and in the presence of tracheoesophageal fistula in the newborn. Aspiration of pus from a purulent sinusitis is common in children and adults alike The aspiration of a piece of tonsil tissue during tonsillectomy in children under general anesthesia is not unknown. The aspiration of a foreign body, most frequently observed in children is particularly harmful when the material is of vegetable source the usual offender is a peanut. The antecedent diseases which most frequently lead to pulmonary abscess are a pre-existent pneumonia and inadequately treated bronchiectasis Necrosis in the center of a large carcinoma, with superimposed infection is not infrequently the first sign that a malignancy exists This is most common in the epidermoid (squamous) type of bronchogenic carcinoma.

Pathology

The pathology of pulmonary abscess is not unlike that of an abscess elsewhere in the body If the defenses of the body are mobilized early and well the abscess is contained by a pyogenic membrane, surrounded by an area of pneumonitis If the abscess is not contained, it may rupture into a bronchus with the discharge of foul material into the trachea, from whence it is coughed up or it may rupture through the visceral pleura resulting in an empyema. Abscesses may occur anywhere in the lung however they are more frequently located at the periphery than centrally

Diagnosis

The diagnosis of pulmonary abscess is usually not difficult to make Symptomatology may begin gradually after the chronicity of previous disease, particularly bronchiectasis or pneumonia. On the other hand, a pulmonary abscess may be silent for a long time and symptoms suddenly appear after a presumed "cure" of antecedent disease There are systemic symptoms and signs due to the infection these include bouts of fever leukocytosis, and general toxemia. The local signs and symptoms depend on the position of the abscess within the lung and include the coughing up of purulent material, bouts of intractable cough pain of pleural origin and hemoptysis Roentgen-ray examination usually reveals the mass and sometimes the fluid level within it (Fig 35) Bronchograms frequently visualize the abscess cavity where plain roentgenography fails to do so Tomography frequently reveals a small abscess cavity in a lesion that plain roentgenography cannot even pinpoint as a space-occupying



FIG 35 Pulmonary abscess in the lower lobe of the right lung of a seven-month-old girl. Note the very thick pyogenic membrane surrounding the accumulation of fluid and gas. Right lower lobectomy Pathologic diagnosis abscess of congenital pneumatocele

lesion Examination of the sputum for the identification of pus and specific bacteria must always be made

Differential Diagnosis

In the differential diagnosis of pulmonary abscess one must consider carcinoma of the lung tuberculosis bronchiectasis empyema, subphrenic pathology congenital pulmonary cysts, fungus disease and echinococcus cysts.

Treatment

The therapy of pulmonary abscess falls logically into its prophylactic and direct phases The prophylactic treatment of pulmonary abscess is that of the antecedent diseases of bronchiectasis and pneumonia plus the meticulous avoidance of aspiration of foreign material especially vomitus during periods of unconsciousness. The direct therapy of lung abscesses falls sharply into acute and chronic phases When an acute abscess is detected every effort must be made to avoid chronicity The use of appropriate antibiotics will aid localization The abscess may then be completely absorbed or may be drained bronchoscopically or percutaneously if the abscess is located subpleurally In the latter case preliminary chemical irritation of the parietal pleura (with such materials as iodoform gauze) will cause the area around the abscess to be walled off from the free pleura the abscess can then be drained to the outside without fear of contaminating the pleural space When a pulmonary abscess becomes large and chronic, definitive operative therapy is mandatory Under cover of appropriate antibiotics open thoracotomy is performed, and the abscess with its area of surrounding pneumonitis, is resected

LESS COMMON PULMONARY INFECTIONS

Fungi

Fungi are frequently recovered in bacteriologic study of pulmonary infections Very frequently they are secondary invaders and are not pathogenic occasionally they may be the organisms causative of disease

Pulmonary actinomycosis is usually secondary to a pre-existing pulmonary infection and may flourish if anaerobic conditions exist in lung tissue Its causative organism is found frequently in the mouth of healthy humans. It must be suspected whenever bronchopulmonary suppuration becomes chronic There is little that is characteristic of pulmonary actinomycosis that is not seen in chronic pulmonary suppurative disease in general In late stages cavitation and tissue destruction are characteristic Identification of the ray fungus is made by microscopic examination of exudate and best of all, by culture Sulfonamides and antibiotics are used in treatment

Coccidiomycosis is produced by the inhalation of its causative organism, *Coccidioides immitis* This fungus is found in the air in some localities of the world more than in others such as the San Joaquin Valley in the United States The largest percentage of cases of pulmonary coccidiomycosis result in a localized pneumonitis which then becomes calcified Occasionally a granuloma is formed and less frequently the disease becomes widely disseminated The diagnosis is made by histologic examination of the sputum, a positive skin test, and a positive blood complement-fixation test. There is no effective therapy

Bronchopulmonary moniliasis an infection with *Candida albicans* is usually secondary to other pulmonary infections Its causative organism is a common saprophyte in the mouth pharynx, gastrointestinal tract, and vagina of healthy human beings Pulmonary moniliasis is seen most commonly as a result of the overgrowth of its causative organism when competing bacteria have been subdued by modern antibiotics The diagnosis is made by histologic recognition and positive cultures obtained from material aspirated from the tracheobronchial tree Therapy is the discontinuance of antibacterial agents and the use of iodides.

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Surgery of Mediastinal Viscera

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THE HEART

Methods of Study and of Therapy

Modern cardiovascular surgery a giant 10 to 15 years of age is the most exciting and dynamic therapeutic development of the current era Many congenital and acquired defects of the heart and great vessels which over the ages spelled invalidism and early death are now being corrected or ameliorated in operating theaters throughout the world Newer technics constantly add to the number of lesions of the heart and aorta which are amenable to surgical attack. In attaining these developments surgeons have applied the methods of the laboratory basing their clinical operations on established physiologic facts testing their procedures

experimentally and then transferring them to their patients The ability of today s surgeon to correct the pathologic physiology of these diseases is not alone a technical achievement it is largely due to a better understanding of cardiorespiratory physiology and its application in the operating room and in the pre and postoperative management of the patient Important contributions by other members of the team including the physiologist anesthesiologist internist and technician have made these therapeutic achievements possible Finally anatomic studies have aided our newer physiologic knowledge in the classification of defects and in the correction of diagnostic errors This is made possible by the detailed pathologic study of surgical and autopsy specimens whenever available It should be pointed out that the most complete and comprehensive classification of ventricular septal defects was made by the pathologist Rokitansky over 150 years ago

More accurate and definitive diagnostic methods have constituted an essential prerequisite both experimentally and clinically and they continue to be evolved. Close attention to history with a detailed physical examination has not been replaced by any laboratory procedures, but rather it always precedes them and is still of paramount importance Newer methods of roentgenographic examination of the chest have been added to the clinician s armamentarium particularly multiple oblique views Multiple lead electrocardiography is frequently of great help *Cardiac catheterization* although not always necessary for diagnosis, is sometimes of inestimable value and frequently provides a pinpoint diagnosis. By this technic not only are pressures in the various chambers and vessels recorded but blood samples disclose differential oxygen contents which help to establish the presence or absence of shunts. *Angiocardiography* the visualization of the passage of blood serially through the great vessels and the chambers of the heart by the use of dyes (2 to 32 frames per second) is occasionally of critically diagnostic value Dye dilution technics are of great additional value in recognition of the presence and size of some arteriovenous shunts

Technics of therapy are continually being evolved and improved Cardiac operations are performed blind under direct vision with an open heart or by semiblind semiopen technics Almost always therapy is preceded by digital exploration, both to confirm the preoperative laboratory findings and to establish the diagnosis if the preoperative findings cannot be confirmed Intracardiac lesions are ideally corrected by direct visualization of the defects with an open preferably dry and quiet, heart. This is accomplished through the technics of *extracorporeal circulation* which involves the removal of all blood flowing to the heart via the venae cavae pumping it (artificial heart) through an artificial oxygenator (artificial lung) and then returning it to the arterial circulation distal to the heart With such a cardiopulmonary bypass the heart can be stilled by drugs, its wall opened, and defects in its interior mechanically corrected Elective cardiac arrest (cardioplegia) was originally produced either with the use of an excess of potassium ion or by the injection into the coronary circulation of acetylcholine more recently *selective hypothermia* has become the method of choice Under these conditions operative time is limited, not by the condition of the stilled heart but by the ability of the patient s blood to withstand the physical and chemical effects of churning in the artificial heart and lung by the viability of the central nervous system during the conditions of bypass and by the ability with the aid of adjunctive procedures to avoid metabolic acidosis.

Open heart work may be accomplished with the aid of systemic hypothermia, permitting inflow occlusion and 3 to 4 minutes of intracardiac manipulation hypothermia may be used as an adjunct during extracorporeal circulation. When no interruption of cardiac function is necessary for the correction of a cardiovascular defect, the oper-

ation may be performed without inflow occlusion and under normothermia. Temporary shunts may be used to bypass the origin of specific great vessels while the latter are repaired or replaced.

Congenital Diseases—General Statement and Classification

Cardiac malformations occur before the end of the tenth week of embryonic life since the development of the heart is virtually complete by that time Numerous factors have been adduced in the explanation of cardiac malformations, but most of these are conjectural in nature It is probable that the occurrence of certain virus diseases in the mother play a role but both the genetics and chemistry of these congenital defects are obscure

Cardiorespiratory physiology of the embryo differs from that of the newborn. In utero placental blood returns by way of the umbilical veins through the ductus venosus thence into the inferior vena cava, through the foramen ovale into the left side of the heart. Superior vena cava return enters the right auricle thence through the tricuspid valve into the right ventricle and out into the pulmonary artery Little blood goes to the lungs, since they are atelectatic. The blood from the pulmonary artery is shunted through the patent ductus arteriosus into the aorta. At birth the lungs begin to expand, they offer a diminished resistance to flow of blood from the right ventricle and the ductus arteriosus normally begins to close

There are certain important and sometimes cardinal signs in congenital heart disease *Cyanosis* or bluish discoloration of the skin and mucous membranes, is an important sign which is present in certain cardiovascular anomalies and absent in others It may be due at least partially to a shunting of venous blood into the systemic arterial circulation through some type of defect The cyanosis may occasionally be subclinical but usually it is detectable in the nail beds in the tips of the ears and the nose and in the mucous membranes and wherever else there are extensive superficial capillary beds Normally blood is 95 per cent saturated with oxygen Not every case of subnormal oxygen saturation of the blood is clinically recognizable as cyanosis. Cyanosis becomes clinically apparent only when 5 or more grams of unoxygenated hemoglobin per 100 ml of circulating blood are present in the circulation at capillary level. In the diagnosis of cyanosis, causes other than cardiovascular defects must be excluded Such extracardiac reasons include respiratory depression, interference with ventilation, and malformation of the lung.

Dyspnea particularly *tachypnea* is a regular companion to cyanosis *Clubbing* of the fingers and toes always accompanies any chronic cyanosis. In the newborn infant it is rarely seen until cyanosis has existed for at least 6 months *Murmurs* are abnormal sounds heard on auscultation They are due to vibrations and eddies in the turbulent blood flow through the chambers and their valves. Areas of abnormal stenosis or areas of sudden drop in pressure due to large septal defects produce variations in the rate of blood flow which set up vibrations giving rise to the abnormal auscultatory findings. (It must be remembered that a large percentage of newborn infants and small children have murmurs which are not dynamically significant and which must be differentiated from those of congenital defects) It is important to determine the point of maximum intensity and the time in the cardiac cycle at which a murmur is heard The grading of intensity and character of murmurs is a matter of personal experience

Enlargement of the heart, and whether it is predominantly of the right or the left side is detected by physical examination by multiple view teleoroentgenography and by electrocardiography *Polycythemia* is a compensatory attempt by the bone marrow to provide more cells to carry oxygen *Syncope* is not uncommon in cyanotic children and is the result of transitory cerebral is

chemia Repeated *pulmonary infections* are not uncommon in the child with congenital heart disease and are usually due to infection superimposed on *pulmonary edema* The symptom of *squatting* is a compensatory method to increase venous return to the heart and thus to increase the blood supply to the lungs *Stunting of growth* is characteristic where there is a large left-to-right shunt. Increased *fatiguability* is a sign of diminished cardiac output *Left heart failure* may first be manifested by dyspnea and pulmonary edema *Right heart failure* is reflected in ascites and tissue edema.

Patent Ductus Arteriosus. The ductus arteriosus (Botallo s duct) is of dynamic significance in intrauterine life in that the blood from the right side of the heart by passes the lung. In extrauterine life this fistula becomes functionally closed within minutes to hours after birth It becomes anatomically obliterated in weeks to a few months after birth and is found only as a ligamentous band between the pulmonary artery and the aorta. Failure of this fistula to close, *patent ductus arteriosus* results in certain circulatory disturbances Since it is a communication between a high-pressure system (aorta) and a low pressure system (pulmonary artery) the shunt of blood is from the aorta to the pulmonary artery In diastole there may be some retrograde flow from the pulmonary artery toward the aorta.

The characteristic dynamic changes in a patent ductus arteriosus are those of an arteriovenous fistula. These include (1) a diminished diastolic pressure resulting in a wide pulse pressure (2) an increased circulating volume (3) hypertrophy and ultimately dilatation of the heart, predominantly of the left ventricle The diagnosis is established on the presence of dyspnea in an underdeveloped child with the characteristic to-and-fro machinery murmur similar to that in any arteriovenous fistula. A thrill may be felt in the area where the auscultatory findings are most pronounced, in the second left intercostal space On fluoroscopy the "hilar dance" is observed and is due to the intermittent transmission of the higher systemic pressure into the branches of the pulmonary artery Electrocardiography will reveal either normal findings or a left axis shift Angiocardiography and cardiac catheterization are seldom necessary in the diagnosis of this defect but they are definitive when the diagnosis is otherwise questionable As long as the heart is competent, there is no cyanosis associated with this malformation The defect may be asymptomatic and may come to notice only with the onset of a subacute bacterial endarteritis

Surgical therapy of patent ductus arteriosus is curative in that normal dynamic relationships are re-established The closure is accomplished either by multiple ligation of the ductus or by division and suture of the ends, the choice being made at the time of operation, depending upon the type and shape of the fistula.

There is one type of patent ductus arteriosus in which surgical therapy is contraindicated This is in the presence of a *reverse shunt* i e when the flow of blood is predominantly from the pulmonary artery to the aorta, a condition which exists together with congenital pulmonary hypertension. Closure of the ductus under such conditions leads to rapid right ventricular dilatation. This problem remains to be solved by a fuller understanding of the cause and therapy of congenital pulmonary vascular disease

Septal Defects Auricular Failure of closure of the septum between the two atria may be at the level of the ostium secundum the latter is cephalad to the former When such a defect occurs there is a shunt from the higher pressure left, side of the heart toward the lower-pressure, right, side Under such conditions some of the blood volume is recirculated more than once through the pulmonary circuit, overloading the latter In some extreme cases pulmonary blood flow may exceed systemic blood flow by several times In the extreme degrees of malformation of the ostium primum there is a failure of the ventricular septum as well as of the

atrial septum Under such conditions there is a common channel between the ventricles and the auricles, known as *atrioventricularis communis*

The diagnosis of atrial septal defect is made on a variety of auscultatory findings an enlarged pulmonary artery, and sometimes an enlargement of the chambers of the right side of the heart Electrocardiographically a right bundle branch block is common this is due to overdistention of the right ventricle during diastole The diagnosis is further confirmed by cardiac catheterization particularly with gas analysis One must be careful in the interpretation of the data in the passage of a catheter from the right to the left side of the heart to be sure that the catheter has not gone through a probe-patent foramen ovale which is normal. In atrioventricularis communis the shunt is maximal, and pulmonary hypertension is the rule

Surgical therapy of atrial septal defects has taken many forms Originally the closed technics (*atrioseptopexy*) were used a blind closure of the septal defect by invagination of the redundant right auricular wall Closure of the defect by a circumferential purse string placed around the defect in the interauricular septum is still applicable in many cases. Most cases at this time are corrected by open heart technics employing either hypothermia or preferably extracorporeal circulation

Ventricular The most common ventricular septal defect is in the membranous portion near the left ventricular outflow tract less common are fenestrations in the muscular septum near the apex. These defects may vary from the smaller ones, which are dynamically insignificant, to the very large ones in which the dynamic changes of the left-to-right shunt are serious In the presence of large ventricular septal defects a gradually increasing pulmonary arteriolar resistance may occur owing to progressive narrowing of the arterial lumina with corresponding pulmonary hypertension, eventuating in a reversal of the shunt from right to left. This results in cyanosis, and the combination of these defects is known as the *Eisenmenger complex* Ventricular septal defects may occur in association with other cardiac defects, such as pulmonic valvular stenosis Occasionally the ventricular septum is entirely absent, resulting in a single ventricle, known as the *trichambered heart*

The diagnosis of ventricular septal defect is usually made from the murmur found to the left of the sternum, which varies in intensity It is greater when the pulmonary resistance is least. If the shunt is large there is frequently a palpable thrill The heart is enlarged in the presence of a large shunt. Diagnosis is further aided by the presence of increased pulmonary vascular markings roentgenographically If there is an associated pulmonary hypertension the right ventricle is enlarged. The electrocardiogram reflects the muscular hypertrophy of whichever chamber is involved. Corroborative evidence is obtained with angiocardiography and cardiac catheterization but these aids are not always necessary

Surgical closure of ventricular septal defects is most satisfactorily accomplished with the aid of extracorporeal circulation, which permits direct visualization and repair by sutures. Small ventricular defects which are not dynamically significant need not be repaired The septal defect associated with Eisenmenger complex may not be closed since its closure would result in further acute right heart strain. Surgical closure of ventricular septal defects either by direct suture or by the placement of a patch of plastic material is sometimes complicated by damage to the bundle of His This may result in a dissociation of the auricular and ventricular contractions, producing a slow ventricular rate Cerebral ischemia may follow if cardiac output is markedly reduced This sequel to ventricular septal defect correction must be avoided and, if it occurs must be treated by re

moval and replacement of sutures, the heart rate must be maintained with the aid of an artificial pacemaker

Pulmonic Stenosis Isolated pulmonic valvular stenosis is the failure of the pulmonic valve to be cleft, the result being a conelike fusion of the valves with a very small orifice The resistance to flow from the right ventricular outflow tract results in a dilatation of this tract there is similarly a poststenotic dilatation of the pulmonary artery due to the jet of blood through the small opening Pulmonic stenosis of the infundibular type, a concentric muscular hypertrophy of the right ventricular outflow tract in the region of the *crista supraventricularis* is usually associated with a high ventricular septal defect.

Pathologic physiology of isolated pulmonic valvular stenosis varies with the degree of right ventricular outflow blockage Interference with normal pulmonary blood flow results in dyspnea, fatiguability and cyanosis upon exertion. Diagnosis is further aided by the rough systolic murmur heard over the pulmonic area, with little or no second pulmonic sound. A thrill is occasionally palpable at this site Roentgenographically the lung fields appear clear The right ventricular preponderance is demonstrated electrocardiographically If there is any doubt about the diagnosis angiocardiography and cardiac catheterization are definitive Surgical therapy of isolated pulmonic valvular stenosis usually produces a spectacular cure The stenosed valve may be opened by a blind transventricular approach or in the more difficult cases under direct vision with the aid of hypothermia or extracorporeal circulation.

Tetralogy of Fallot. The combined defects known as the *tetralogy of Fallot* constitute the most common cyanotic congenital heart disease The pathologic findings are those of (1) a high ventricular septal defect (2) a pulmonic stenosis, usually of the infundibular type although it may be of the valvular type (3) some degree of overriding of the aorta so that this vessel receives some blood from the right ventricle resulting in (4) right ventricular hypertrophy If the pulmonic stenosis is complete constituting *atresia of the infundibulum* the only blood going to the lungs will be through the bronchial arteries plus that which reaches the lungs through the ductus arteriosus which is frequently patent under these conditions. Such a situation constitutes a *pseudotruncus* Right aortic arch is not uncommon in the presence of tetralogy An associated right to-left shunt through a patent foramen ovale converts the tetralogy into a pentalogy

Diagnosis is aided by the association of impaired physical development in addition to the cyanosis the phenomenon of squatting for the relief of dyspnea and a "sternal heave" because of the right ventricular hypertrophy Roentgenographically the right aortic arch may be visualized, the peripheral lung fields are clear and vascular pulsations are minimal. The convexity which usually represents the pulmonary artery is replaced by a concavity Electrocardiographically the right ventricular hypertrophy is evident. Cardiac catheterization and angiocardiography are very useful in that they may reveal the pattern of the intracardiac circulation and the direction of the shunt.

The therapy of the tetralogy of Fallot leaves much to be desired Yet the early interest in this disease and the original development of an operation for its amelioration, by Blalock and Taussig, served as a stimulus for the entire field of cardiac surgery The original operation consisted of shunting systemic blood from a subclavian artery to a branch of the pulmonary artery This effect was achieved in infants by the Potts operation of side-to-side anastomosis of the aorta with the left pulmonary artery These operations are palliative procedures only in that they bring more blood to the lungs, bypassing the pulmonic stenosis by the newly created A-V fistula. More recently more definitive corrective technics

are available with the use of extracorporeal circulation. The infundibular or valvular stenosis can be corrected as well as closure of the ventricular septal defect. Although not yet as uniformly safe as the latter operation this approach seems to offer more in a definitive manner

Rare and Combined Lesions. Aortic-Pulmonary Septal Defect (Aortic Pulmonary Window) This defect is a communication between the aorta and the main pulmonary artery just distal to their respective valve outlets It is essentially a side-to-side fistula between these two great vessels, and the pathologic physiology is exactly that of a patent ductus arteriosus, i e., an arteriovenous fistula. The differential diagnosis between a ductus and an aortic pulmonary septal defect is made essentially by catheterization and angiocardiography Because of the precarious position of this defect it can be corrected with safety only with the use of extracorporeal circulation technics.

Aortic Stenosis Congenital aortic stenosis (valvular and subvalvular) is among the less common congenital defects The diagnosis is usually not difficult, characteristic are the systolic murmur and thrill and left ventricular hypertrophy The thrill is transmitted cephalad along the course of the great arteries of the neck.

The treatment of congenital valvular aortic stenosis does not differ from that of the acquired variety which is described later Subvalvular stenosis is not usually dynamically significant, although the auscultatory findings may be maximal.

Tricuspid Atresia Tricuspid atresia is another form of cyanotic congenital heart disease It must be associated with a patent foramen ovale or an atrial septal defect to be compatible with life These pathways permit blood to pass from the right to the left atrium the resulting right-to-left shunt is the reason for the cyanosis Blood is supplied to the lungs by way of the bronchial arteries or possibly through a concomitant patent ductus arteriosus Tricuspid atresia is frequently associated with hypoplasia of the pulmonary arteries The diagnosis is made by the finding of bulging and pulsating jugular veins in the neck and a pulsating liver Roentgenographically a right aortic arch is frequently seen, and the area of the pulmonary artery is concave. Electrocardiographically there is a marked left ventricular preponderance, atrial hypertrophy is also signified by large P waves Angiocardiography is definitive Therapy is much the same as in the cases of tetralogy consisting in shunting of blood from a systemic artery to a pulmonary artery

Vascular Rings These anomalies are usually associated with a normal heart. The most common type of vascular ring is the persistent double aortic arch which encircles the mediastinal viscera. Symptoms are produced by the compression of either the trachea or the esophagus or both The large vessels to the upper extremity or to the head, the subclavian and carotids may have anomalous origins and may course over the trachea and esophagus in a manner which obstructs them

The diagnosis is suspected in an infant who has difficulty in breathing or in feeding Respiratory stridor and/or dysphagia are characteristic The diagnosis is made by the roentgenographic examination of the mediastinal viscera which are compressed Barium swallow may lead to a positive diagnosis

Surgery is indicated as soon as the diagnosis is made and consists in division of the lesser ring more recently in addition the anomalously placed vessels are severed and restored to a normal position, with the aid of a graft if necessary

Persistent Truncus Arteriosus The persistence of a single trunk arising from both ventricles is a severe congenital cyanotic heart deformity Several types are found some of which cannot be differentiated clinically from the tetralogy of Fallot Usually the lungs receive their blood supply from hypoplastic pulmonary arteries arising from the single aorta " occasionally only from bronchial vasculature Treatment consists

in shunting arterial blood to the lung by the Blalock or Potts procedure and is only palliative to a limited degree

Transpositions. This severe malformation as the name implies, consists in the aorta originating from the right ventricle and the pulmonary artery from the left ventricle It is associated with cyanosis from birth Extrauterine life is not possible unless there are associated communications between the two circulations, with bidirectional mixing Angiocardiography reveals the defects in the great vessels described above

Therapy of this severe defect has been difficult. Retransposition of the vessels as they emerge from the heart has not been successful, essentially because the coronary arteries cannot be transposed together with the aorta. The most successful technic to date is the operation of Baffes in which venous transposition is accomplished In this procedure the pulmonary venous return from the right lung is directed into the right atrium, and the inferior vena cava is directed into the left atrium by means of an arterial graft. By this means both ventricles contain mixed blood and relative oxygenation is attained.

Acquired Diseases

Rheumatic Fever Heart Disease When the heart is involved during the acute phase of rheumatic fever it is usually in the form of a pancarditis with evidence of pericarditis myocarditis, and endocarditus The pericarditis may or may not be associated with effusion Late dynamic sequelae of the pericarditis are uncommon The myocarditis can be detected by electrocardiographic changes, but more importantly by the evidence of cardiac decompensation The typical lesion found in the myocardium is the Aschoff nodule which has become synonymous with rheumatic fever activity The myocardial involvement may lead to chronic muscle impairment and congestive failure

The endocardial involvement of the valves is recognized by changes in the heart sounds or murmurs which are characteristic of one or another valvular defect. In the active stage of the disease vegetations are formed on the endocardial surfaces of the valves which become vascularized as the healing progresses This granulation tissue progresses to cicatrization with resulting distortion of the valves and of the chordae tendineae, leading to corresponding impairment of function. The late dynamic results of the endocarditis then are due to changes in the structure and function of the valves While the mitral is the valve most frequently affected, all other valves alone or in combination, may be involved If the mitral valve is scarred and cannot close completely during ventricular systole a portion of the blood ejected with each left ventricular contraction is regurgitated into the left atrium, constituting mitral regurgitation (leakage) When the commissures of the mitral valves and their cusp edges become involved in the healing process the aperture is narrowed and the valves are unable to open during ventricular diastole preventing blood from entering the ventricle freely from the left atrium This is mitral stenosis. The valves, commissures, and chordae tendineae may be so involved in the scarring process that a combination of regurgitation and stenosis may occur The same cicatricial process may also affect the other valves, producing upon occasion aortic regurgitation and stenosis and tricuspid stenosis.

Rheumatic heart disease comprises about 30 per cent of all clinically encountered heart lesions The methods employed in studying the dynamics of the circulation in these cases are similar to those employed for congenital lesions. Usually but not always a history of antecedent rheumatic activity is obtained, most commonly during childhood The findings on physical examination and the laboratory data available from such procedures as roentgenography angiocardiography and cardiac catheterization will be discussed with each lesion The surgical therapy of rheumatic lesions has in the past been accomplished, for the most part, with

mechanically correctable congenital lesions

Aortic Stenosis The mechanical change in aortic stenosis is the obstruction to the outflow of the left ventricle With the increased work performed by the left ventricle in its attempt to eject a normal blood volume, the left ventricular wall becomes hypertrophied. The reduced output causes periods of ischemic syncope especially on effort. Myocardial ischemia, due to partial occlusion of the coronary ostia, results in angina. This triad of syncope angina, and left ventricular hypertrophy is a constant part of the natural history of this disease Surgical efforts to dilate the aortic valve blindly by a transleft ventricular procedure have met with less success than those directed to the mitral orifice because the approach requires cutting the left ventricle or the aortic wall and also because the aortic valve leaflets do not seem to regain their flexibility More recently the technic of extracorporeal circulation has been extended to patients with aortic stenosis despite which the benefits are still limited because of the thickness and inelasticity of the stenotic valve Correction of the dynamics secondary to this lesion now are directed toward the development of prostheses which will replace the valve This has been difficult of accomplishment as yet because of encroachment of such appliances on the coronary artery ostia

Aortic Regurgitation In aortic regurgitation the size of the left ventricle reaches gigantic proportions (cor bovinum.) The pathologic physiology is the incompetence of the aortic valve in that it no longer acts as a baffle to prevent reflux from the aorta into the left ventricle The work load of the left ventricle is thus greatly increased. No successful technic for the direct correction of the valve has as yet been devised Complete replacement by a satisfactory prosthesis as in the case of aortic stenosis, is a hope for the future Hufnagel and his group have reported the successful use of plastic valves placed in the descending aorta in counteracting the deleterious effects of aortic regurgitation upon the heart. A ball valve baffle is placed in the descending thoracic aorta, which prevents reflux of blood into the left ventricle from about 75 per cent of the body This operation is currently lifesaving for those patients with aortic regurgitation who experience congestive heart failure

Coronary Artery Heart Disease The captain of men of death and one of the greatest scourges of our day is coronary artery heart disease. It is at least twice as frequent as all forms of rheumatic heart disease combined and about thirty times as frequent as all forms of congenital heart disease The basic disease is no different from obliterative arterial disease elsewhere (as discussed in Chapters 11 and 12) Myocardial ischemia is predominantly the sequel to atherosclerosis of the coronary vessels which may be either occluded or narrowed sufficiently that there is a disparity between oxygen demand and supply Such disparity may also occur in cardiomegaly due to other causes, in which even normal vessels are unable to supply a requisite quantity of oxygen. Ischemia may result in infarction or in diffuse fibrosis Infarction is usually but not invariably the end result of occlusion of a coronary artery The left ventricle specifically is sensitive to relative anoxia. It is widely thought today that atherosclerosis may be an expression of specific disturbances in the metabolism of cholesterol and fat, under the stimulus of influences not yet clearly defined The ultimate solution to this grave problem would seem to depend upon a better understanding of the metabolism of foodstuffs and of degenerative processes in general

Pathology The pathologic physiology of chronic coronary insufficiency is essentially that of an obliterative endarteritis. Plaques of fatty material are deposited on the intima of the coronary arteries (as elsewhere) and superimposed on these roughened surfaces, thromboses occur When fine branches of the coronary arteries are thus involved small areas of myocardial ischemia and fibrosis result Many such areas may occur and be

compatible with survival. Occasionally however a small "dry" area may act as a trigger mechanism (electrical instability of the myocardium) and lead to sudden, rapid death from ventricular fibrillation or asystole If a major branch is occluded, recovery is possible but sudden death is altogether too common

The diagnosis of coronary artery heart disease is based upon the manifestations of myocardial ischemia. The degree and location as well as the progress of infarction may be traced electrocardiographically by repeated examinations. The major sequelae of myocardial infarction are *embolic phenomena congestive heart failure* and *aneurysmal dilatation* of the ventricular wall.

Medical therapy of acute and chronic coronary insufficiency resolves itself into the prevention of congestive heart failure by rest and the avoidance of the deleterious effects of arrhythmias by pharmacologic means It is doubtful at the present time whether it alters the ravages of this disease although an attempt to avoid hyperlipemia and hypercholesterolemia is directed toward this end. Anticoagulants have been found helpful in both the acute and chronic stages.

Surgical Therapy Because of the unabated serious mortality and morbidity associated with chronic coronary insufficiency surgical approaches to this problem constitute one of medicine s most pressing problems. Many efforts are being expended in this direction with encouraging, but not completely unequivocal, results Recognition should be given to the work of Claude Beck, who has devoted the greater part of his active life toward the development of surgical methods for revascularizing the ischemic human heart. Surgical therapy of coronary artery heart disease is directed toward restoring arterial circulation of the myocardium and preventing ventricular fibrillation should subsequent occlusions occur and preventing the sequelae of infarction It aims to do this by bringing a greater supply of oxygen-carrying blood to the heart muscle. If this can be accomplished surgically it leads to the alleviation of the pain of *status anginosus* the diminution in size of any future infarct subsequent to the occlusion of a coronary vessel, and the prevention of immediate death by dysrhythmia in subsequent coronary occlusion

Efforts toward the relief of chronic coronary insufficiency by bringing an increased supply of blood to the myocardium have followed two directions An ischemic area of myocardium can be revascularized via an extracoronary source of blood or by a redistribution to a dry area of blood which has already entered existing patent coronary arteries The development of intercoronary anastomoses is accomplished by the ligation of the coronary sinus as first practiced clinically by Beck. Experimental animals and patients subjected to this procedure exhibit new collateral channels between the existing patent coronary arteries. In addition, patients so treated have in large measure, been relieved of anginal pain, have been able to return to their previous occupations, and have been protected through subsequent occlusive episodes Accompanying the procedure of coronary sinus ligation may be the performance of a cardiopericardiopexy with various substances the epicarditis so produced enhances the development of intercoronary channels on the surface of the heart.

Operations designed to bring an extracoronary source of blood to the myocardium have also been tried and favorably reported. These procedures consist essentially in implanting the end of a patent peripheral artery into the myocardium. Thromboendarterectomy of an occluded coronary artery has been suggested and clinically applied the limitations of such a procedure are great, as they are in the peripheral circulation. It is conceivable that, with the application of extracorporeal circulation technics blood vessels in the heart may be replaced as they are peripherally

Aneurysm The weakening of an ischemic area of ventricular myocardium with the

mechanically correctable congenital lesions.

Aortic Stenosis The mechanical change in aortic stenosis is the obstruction to the outflow of the left ventricle With the increased work performed by the left ventricle in its attempt to eject a normal blood volume the left ventricular wall becomes hypertrophied. The reduced output causes periods of ischemic syncope especially on effort Myocardial ischemia, due to partial occlusion of the coronary ostia, results in angina. This triad of syncope angina, and left ventricular hypertrophy is a constant part of the natural history of this disease Surgical efforts to dilate the aortic valve blindly by a transleft ventricular procedure have met with less success than those directed to the mitral orifice because the approach requires cutting the left ventricle or the aortic wall and also because the aortic valve leaflets do not seem to regain their flexibility More recently the technic of extracorporeal circulation has been extended to patients with aortic stenosis despite which the benefits are still limited because of the thickness and inelasticity of the stenotic valve Correction of the dynamics secondary to this lesion now are directed toward the development of prostheses which will replace the valve. This has been difficult of accomplishment as yet, because of encroachment of such appliances on the coronary artery ostia.

Aortic Regurgitation In aortic regurgitation the size of the left ventricle reaches gigantic proportions (cor bovinum.) The pathologic physiology is the incompetence of the aortic valve in that it no longer acts as a baffle to prevent reflux from the aorta into the left ventricle The work load of the left ventricle is thus greatly increased No successful technic for the direct correction of the valve has as yet been devised Complete replacement by a satisfactory prosthesis as in the case of aortic stenosis, is a hope for the future Hufnagel and his group have reported the successful use of plastic valves placed in the descending aorta in counter acting the deleterious effects of aortic regurgitation upon the heart. A ball valve baffle is placed in the descending thoracic aorta, which prevents reflux of blood into the left ventricle from about 75 per cent of the body This operation is currently lifesaving for those patients with aortic regurgitation who experience congestive heart failure

Coronary Artery Heart Disease. The captain of men of death and one of the greatest scourges of our day is coronary artery heart disease It is at least twice as frequent as all forms of rheumatic heart disease combined and about thirty times as frequent as all forms of congenital heart disease The basic disease is no different from obliterative arterial disease elsewhere (as discussed in Chapters 11 and 12) Myocardial ischemia is predominantly the sequel to atherosclerosis of the coronary vessels which may be either occluded or narrowed sufficiently that there is a disparity between oxygen demand and supply Such disparity may also occur in cardiomegaly due to other causes, in which even normal vessels are unable to supply a requisite quantity of oxygen. Ischemia may result in infarction or in diffuse fibrosis Infarction is usually but not invariably the end result of occlusion of a coronary artery The left ventricle specifically is sensitive to relative anoxia. It is widely thought today that atherosclerosis may be an expression of specific disturbances in the metabolism of cholesterol and fat, under the stimulus of influences not yet clearly defined. The ultimate solution to this grave problem would seem to depend upon a better understanding of the metabolism of foodstuffs and of degenerative processes in general

Pathology The pathologic physiology of chronic coronary insufficiency is essentially that of an obliterative endarteritis Plaques of fatty material are deposited on the intima of the coronary arteries (as elsewhere) and superimposed on these roughened surfaces, thromboses occur When fine branches of the coronary arteries are thus involved small areas of myocardial ischemia and fibrosis result Many such areas may occur and be

compatible with survival Occasionally however a small "dry" area may act as a trigger mechanism (electrical instability of the myocardium) and lead to sudden rapid death from ventricular fibrillation or asystole If a major branch is occluded, recovery is possible but sudden death is altogether too common

The diagnosis of coronary artery heart disease is based upon the manifestations of myocardial ischemia The degree and location, as well as the progress of infarction may be traced electrocardiographically by repeated examinations The major sequelae of myocardial infarction are *embolic phenomena congestive heart failure* and *aneurysmal dilatation* of the ventricular wall

Medical therapy of acute and chronic coronary insufficiency resolves itself into the prevention of congestive heart failure by rest and the avoidance of the deleterious effects of arrhythmias by pharmacologic means It is doubtful at the present time whether it alters the ravages of this disease although an attempt to avoid hyperlipemia and hypercholesterolemia is directed toward this end. Anticoagulants have been found helpful in both the acute and chronic stages

Surgical Therapy Because of the unabated serious mortality and morbidity associated with chronic coronary insufficiency surgical approaches to this problem constitute one of medicine's most pressing problems Many efforts are being expended in this direction with encouraging, but not completely unequivocal, results Recognition should be given to the work of Claude Beck, who has devoted the greater part of his active life toward the development of surgical methods for revascularizing the ischemic human heart Surgical therapy of coronary artery heart disease is directed toward restoring arterial circulation of the myocardium and preventing ventricular fibrillation should subsequent occlusions occur and preventing the sequelae of infarction. It aims to do this by bringing a greater supply of oxygen-carrying blood to the heart muscle

If this can be accomplished surgically it leads to the alleviation of the pain of *status anginosus* the diminution in size of any future infarct subsequent to the occlusion of a coronary vessel and the prevention of immediate death by dysrhythmia in subsequent coronary occlusion

Efforts toward the relief of chronic coronary insufficiency by bringing an increased supply of blood to the myocardium have followed two directions An ischemic area of myocardium can be revascularized via an extracoronary source of blood or by a redistribution to a dry area of blood which has already entered existing patent coronary arteries The development of intercoronary anastomoses is accomplished by the ligation of the coronary sinus as first practiced clinically by Beck. Experimental animals and patients subjected to this procedure exhibit new collateral channels between the existing patent coronary arteries. In addition, patients so treated have in large measure been relieved of anginal pain, have been able to return to their previous occupations, and have been protected through subsequent occlusive episodes Accompanying the procedure of coronary sinus ligation may be the performance of a cardiopericardiopexy with various substances the epicarditis so produced enhances the development of intercoronary channels on the surface of the heart.

Operations designed to bring an extracoronary source of blood to the myocardium have also been tried and favorably reported These procedures consist essentially in implanting the end of a patent peripheral artery into the myocardium. Thromboendarterectomy of an occluded coronary artery has been suggested and clinically applied the limitations of such a procedure are great, as they are in the peripheral circulation. It is conceivable that, with the application of extracorporeal circulation technics, blood vessels in the heart may be replaced as they are peripherally

Aneurysm The weakening of an ischemic area of ventricular myocardium with the

formation of an aneurysm has long been known as a late sequel to myocardial infarction In recent years some of these aneurysms have been excised

Until the basic metabolic problems of atherosclerosis are solved, any operative procedures which can be shown to decrease morbidity and mortality of chronic coronary insufficiency and provide longevity are legitimate pursuits

Wounds. Wounds of the heart are inflicted by sharp instruments, high velocity missiles, or blunt trauma to the chest wall. In stabbing by sharp instruments or penetration by high velocity missiles death is frequently rapid, owing to dysrhythmia massive blood loss or sudden severe tamponade This last named term means interference with cardiac function from rapid accumulation of blood within the pericardial sac If the patient reaches the hospital alive with tamponade, immediate aspiration may be lifesaving If the hemopericardium recurs within a short period of time operative intervention is indicated If pericardial aspiration possibly repeated and replacement of lost blood by transfusion leads to progressive improvement, operative intervention may not be necessary

Blunt trauma to the heart, in both automotive and industrial accidents, is more common than usually appreciated. Whenever there is a history or evidence of a blow having been struck on or near the sternum (steering wheel injury) and signs of decompensation, including dyspnea, tachycardia, and peripheral edema, a myocardial contusion must be suspected Serial electrocardiography is corroborative Pathologic examination usually at autopsy reveals gross hemorrhage in the myocardium and possible actual rupture of the muscle Therapy does not differ from that of an acute myocardial infarction

Circulatory Arrest (Cardiorespiratory Failure; Cardiac Arrest)

Etiology and Types. Cardiac arrest is the term usually applied to the acute sudden cessation of an effective heartbeat. It is a general term and is equally applicable to its two types (1) asystole or standstill and (2) ventricular fibrillation In either case there is an arrest of cardiac, and therefore of circulatory function It may constitute a complication during a surgical operation although similar events frequently accompany acute myocardial infarctions and other diseases. It is the ultimate of acute emergencies and is almost always sudden, although there may be premonitory warnings. Many so-called "anesthetic deaths" are really incidents of preventable cardiorespiratory failure The cardinal factor in the causation of cardiac arrest is *hypoxia Vagal reflexes* which slow and inhibit the heart are sometimes partially to blame however it is doubtful whether such reflexes are as noxious in the presence of adequate oxygenation as they are under a state of hypoxia Other factors which contribute to the precipitation of cardiac arrest are hypercapnia (increased carbon dioxide content in the blood) and overdistention of the right heart. Electrocution (which leads to ventricular fibrillation) and suturing of the bundle of His (which produces A V block) also produce circulatory arrest Predisposing factors include primarily pre-existent heart disease especially chronic coronary insufficiency or congestive heart failure Reflexes initiated by traction on autonomic nervous system structures and by shock inducing procedures are additional predisposing factors. It is probable that all these lesser causes in some way are connected with the main factors of hypoxia and vagal reflexes.

The effects of cardiac arrest (cardiorespiratory failure) are generalized anoxia and rapid death The central nervous system is peculiarly susceptible to oxygen deficiency and unless effective circulation is restored within 3 to 4 min permanent damage to both cortical and medullary centers occurs, even though cardiac function may be restored after this interval

Diagnosis. The diagnosis of cardiac arrest must be made immediately by the op-

erating personnel or by the anesthesiologists. The evidence to the surgeon is lack of pulsation or failure of vessels to bleed to the anesthesiologist it is the absence of an effective systemic blood pressure It may be detected outside the operating room when there is sudden disappearance of a peripheral pulse or rapid loss of consciousness

Therapy Prophylactic and Definitive The therapy of circulatory failure falls clearly into its two component parts (1) prophylactic, (2) definitive The cardiac status of all patients should be carefully evaluated before they are subjected to surgical operations to detect diseased states which may be amenable to correction or which may contraindicate elective surgery Hypovolemia due to blood loss must be prevented or corrected by appropriate replacement The occurrence of cardiac arrest will be minimized if meticulous attention is paid to *continuous adequate oxygenation* of a patient under anesthesia Noxious reflexes may be abolished by adequate amounts of atropine in the preoperative period and during operative procedures

When the diagnosis of cardiac arrest is made a state of emergency exists Time is all important. Corrective measures in proper sequence must be initiated immediately These steps constitute the act of *cardiac resuscitation* and include (1) *positive pressure anesthesia* with an indwelling endotracheal tube (2) *immediate thoracotomy* through the fourth or fifth left intercostal space and (3) *maintenance of circulation* by intermittent *manual systole* by compression of the heart (incorrectly called *cardiac massage*) All other procedures can wait until these three essentials are accomplished. If after the pericardium is opened the heart is observed to be in standstill (asystole) intermittent manual compression at a rate of about 60 per minute alone will often suffice to cause the resumption of a cardiac beat. If the ventricle is fibrillating intermittent compression is still effective and must be continued. There is no great urgency about defibrillation as long as the circulation and respiration are maintained An electric defibrillator may then be brought in and applied to the heart until asystole is produced This asystole is then treated as if it had been found in the first place If manual compression fails in the restitution of a normal beat the injection of epinephrine may do so If it does not, several milliliters of 10 per cent calcium chloride are usually effective In each case the drug is injected into the lumen of the left ventricle and is followed by manual compression which results in delivering the drug to the myocardium via its coronary circulation If hypovolemia has not been corrected, it should be done as promptly as possible The *artificial pacemaker* offers great promise It may be substituted for intermittent manual compression of the heart, obviating direct trauma to the myocardium and also avoiding the necessity of opening the chest. It may serve additionally in dysrhythmic states occurring outside of the operating room

The incidence of cardiorespiratory arrest during anesthesia seems to be anywhere from 1 in 1,000 to 1 in 5 000 cases. It may occur during any type of surgery but is more often met with when the heart is handled directly Reports in the literature to date indicate that only about one-fourth of all cases of cardiac arrest are successfully resuscitated. If more than 4 minutes of cardiorespiratory failure have elapsed, the patient may well be little more than a vegetating organism even if circulation is successfully restored When circulatory arrest occurs in an otherwise normal heart owing to any of the above mentioned factors resuscitation is far more often successful than if the arrest occurs in a diseased heart.

THE PERICARDIUM

Surgical Anatomy and Physiology

The pericardial sac, which contains the heart and the origins of the great vessels, consists of two layers, an outer fibrous and an inner serous. The serous lining is re

flected onto the heart as the visceral pericardium (epicardium) The fibrous envelope blends with the central tendon of the diaphragm inferiorly and the roots of the great vessels superiorly The apposed serous surfaces of the pericardium and the epicardium allow for free motion of the heart within the sac and at the same time protect the organ from ready extension of inflammatory mediastinal and pulmonary disease The pericardium aids in fixing the heart within the mediastinum and in restricting its displacement with each beat and with change in body position It enlarges to accommodate gradual increases in size of the heart. However in conditions of acute dilatation of the heart the pericardial envelope may constitute a limiting factor

Effusions

Pericardial effusions may be of hemorrhagic, serous, or pyogenic nature Hemorrhage may be acute or chronic the acute is usually traumatic in origin (tamponade) the chronic due to metastatic malignant disease Serous pericardial effusions are those of congestive heart failure or of inflammatory origin the latter may be either tuberculous or rheumatic Pyogenic effusions are either secondary to trauma, direct extensions from adjacent infections or rarely metastatic in pyemic states

The diagnosis of pericardial effusion is usually made on the basis of the interference with cardiac function The accumulation of fluid diminishes the cardiac inflow and results in the *Beck triad* of (1) an elevated venous pressure (2) a reduced systemic arterial pressure and (3) a small quiet heart on fluoroscopy Therapy discussed in Chapter 17 *consists of aspiration for relief of* tamponade identification of the nature of the fluid and treatment of the underlying disease

Constrictive Pericarditis

During the course of acute pericardial inflammation there is deposition of fibrinous exudate upon the serous surfaces This exudate undergoes organization characterized by the usual invasion of fibroblasts and the conversion of fibrinous deposits and adhesions into mature fibrous tissue These fibrous synechias destroy the smooth, gliding mechanism and increase the load on the heart by freezing it to the unyielding fibrous envelope especially if there is contraction of the heavy fibrous pericardial scar

Compression of the heart occurs when the unyielding pericardial sac contracts sufficiently to impinge upon the heart preventing its adequate filling in diastole and subsequently its adequate ejection in systole. The result is a diminution in stroke volume The inability of the left side of the heart to fill in diastole is productive of pulmonary hypertension. The reduced output of the left ventricle is responsible for the severe dyspnea on exertion Since cardiac output cannot be increased with increasing demand, tachycardia results and venous pressure rises above levels found in congestive heart failure The obstruction to inflow (inflow stasis) is partly due to the pulmonary hypertension and partly to direct constriction of the right atrium and the venae cavae Ultimately a severe passive hyperemia develops, involving the liver (hepatic outflow hypertension) which results in ascites Edema of the peripheral tissues follows. Significant pulmonary hypertension may be demonstrable by cardiac catheterization

The diagnosis of chronic constrictive pericarditis is based on the history of a preexisting disease that may have included pericarditis and it includes the *Beck triad* described above plus added symptoms due to *chronicity dyspnea* and *ascites* Diminished cardiac output and prolonged *circulation time are demonstrable* Electrocardiographic evidence includes reduction in QRS amplitude and primary T wave changes with broad and notched P waves Calcification of the pericardium may be visualized roentgenographically

Surgical therapy consists of *pericardiectomy* or cardiac decortication Removal of the encasement of the heart increases dia

stolic filling of the ventricles reduces the pulmonary hypertension and diminishes the venous pressure with disappearance of ascites and edema.

Tumors

Tumors of the pericardium are uncommon. They are usually of connective tissue origin most commonly lipomas Rarely a malignant neoplasm is described

THE GREAT VESSELS

Congenital Coarctation

Coarctation of the aorta is a congenital malformation in which there is a narrowing of the aortic lumen in the region of the ductus arteriosus All degrees of constriction occur The impediment to blood flow in the aorta results in a large number of collateral vessels which develop to bypass the area of obstruction The vessels involved are those of the neck and shoulder girdle above the obstruction and the intercostal and internal mammary vessels below it. Two main types are found The *infantile type* occurs proximal to a patent ductus arteriosus. Associated cardiac malformations are common The *adult type* of coarctation is found distal to the ductus arteriosus.

Collateral vascular dilatation is not marked with the infantile type because when the ductus is distal to the coarctation there is pulmonary hypertension and the aorta receives most of its blood through the ductus This is the reason for the name *infantile*

Diagnosis is usually made on the physical findings of (1) a high arterial pressure in the upper extremities with a low pressure and peripheral ischemia in the lower extremities (2) inability to palpate femoral pulses and (3) the roentgenographic finding of huge intercostal vessels, which may notch the inferior borders of the ribs The large collaterals in the chest wall frequently give rise to diffuse continuous murmurs. In the infantile type of coarctation there may be cyanosis and clubbing of the toes Cardiomegaly and congestive failure are common Electrocardiography confirms the left ventricular hypertrophy Aortography may define the type and extent of the coarctation

Surgical therapy of coarctation of the aorta is indicated in every case of the so-called adult type with or without a patent ductus arteriosus. In children this is usually possible without the aid of a homograft a graft may and should be used if necessary Operations above the age of twenty are seldom performed Not only are the vessels more difficult to handle but irreversible changes in the cerebral vasculature may already have occurred The infantile type of coarctation may be operated upon in a similar manner except in cases where there is a severe pulmonary hypertension.

Acquired: Aneurysms

The etiology pathology and therapy of arterial aneurysms have been discussed in Chapters 10 and 11 Aortic aneurysms produce symptoms mainly because of pressure on adjacent structures. There are few systemic circulatory disturbances. Arteriosclerotic aneurysms of the aorta are most common in the descending portion and its branches particularly at their bifurcations They are treated by resection and replacement with homografts or appropriate prostheses Luetic aneurysms are more common in the ascending aorta. Only recently has this segment been successfully resected and replaced. This procedure requires the temporary shunting of blood from the heart to the vessels of the head and the descending arch

Dissecting aneurysm is most common in the descending aorta, thoracic and abdominal It occurs to less extent in smaller vessels. The pathology is the dissection of blood between the intima and the media. Entrance to this space is gained through a break in the aortic intima. The combination of hypertension and atherosclerosis usually is required to permit this set of circumstances The dissection may continue to and beyond

the bifurcation of the aorta and may include the renal and mesenteric vessels as well The De Bakey operation has been designed to correct further dissection of blood between the walls of the aorta and to permit re-entry from the false channel into the aortic lumen distally More recently the entire area of dissection has been removed and replaced with an appropriate *homograft or inanimate prosthesis*

THE MEDIASTINAL SPACE

Anatomy

The mediastinum is the central compartment of the chest, and is bounded by the posterior aspect of the sternum ventrally the anterior surface of the vertebral bodies dorsally the two pleuropulmonary areas laterally, and the diaphragm inferiorly The loose tissue of the mediastinum is continuous with that of the cervical region the mediastinal vessels and other viscera are continuous with those of the neck, and the aorta and the esophagus are continued distally

Many vital structures lie within the mediastinum the heart and great vessels the trachea and main bronchi the esophagus, vagus and phrenic nerves and many lymph glands The mediastinum is conventionally considered to be divided into anterior and posterior compartments by a plane passing in front of the tracheal bifurcation. The anterior mediastinum is further divided into superior and inferior portions by a transverse plane passing through the level of the superior border of the heart. The superior division houses the thymus and great vessels the inferior division is occupied by the pericardium and heart. The posterior mediastinum contains the trachea bronchi esophagus aorta, thoracic duct vagus and sympathetic nerves and azygous veins

With good anesthesia this space is now *frequently and extensively explored for the* surgical correction of diseases and the sequelae of accidents and traumas.

Infections

Infections are brought to the mediastinum by the blood or lymph streams or through direct extension from contiguous structures. Chronic infections of the lymph nodes especially by tuberculosis, are common throughout the world. Enlarged lymph nodes in the mediastinum due to chronic infection are sometimes difficult to differentiate from sarcoidosis or lymphomas Infection may descend into the mediastinum along the fascial spaces from the pharynx and neck Direct extension from the pleuropulmonary spaces into the mediastinum is not uncommon. Acute infections of the mediastinal space due to instrumentation of the esophagus and the tracheobronchial tree are more frequent today with the increased use of these methods of study The penetration of a foreign body through the wall of either of these viscera, especially in children is not infrequently seen. Perforation of the esophagus as a result of disease and rupture of the esophagus owing to increased hydrostatic pressure are discussed in detail in Chapter 22

Inflammatory disease of the mediastinal space may result either in abscess formation or more commonly in a diffuse phlegmonous mediastinitis The diagnosis of *mediastinitis* is made on the symptoms and signs of a local infection with systemic sequelae The former include thoracic discomfort and pain possibly dyspnea and dysphagia The systemic signs are those of a violent infection, particularly if a phlegmonous mediastinitis exists.

Therapy follows the general principles of infection of a closed space anywhere in the body Drainage *per mediastinotomy* is appropriate even when there is no positive evidence of the formation of pus This is true because of the nature of this closed space and of the vital structures that it houses. Mediastinotomy may be accomplished *anteriorly with a simple incision in the supra* sternal notch superior mediastinotomy may also be effected through the neck with en-

trance in the tracheoesophageal groove Posterior mediastinotomy through an intercostal space dorsal to the angle of the ribs is usually reserved for those cases in which there is evidence of pus formation Antibiotics are used to limit the spread of the infection and are particularly helpful when the infection is a diffuse phlegmon

Neoplasms, Primary and Metastatic

The common tumor of the anterior and superior mediastinum is the dermoid It is usually symmetrical frequently gives evidence of its nature roentgenographically and is often found on routine chest films Tumors that arise from other embryonic rests are occasionally seen in the same area.

The usual tumor of the posterior mediastinum is a neurofibroma arising from the roots of the intercostal nerves It is smooth in outline and more commonly than not is asymptomatic An occasional neurofibrosarcoma is described

The various types of tumors in the lymphoma group are most troublesome both in diagnosis and in therapy These tumors are multicentric in origin and are described in Chapter 14

An enlarged thymus is occasionally responsible for a shadow in the mediastinum which is erroneously taken for a tumor Very little is known about the physiology of this organ Tumors of the thymus are frequently found in association with myasthenia gravis

Wounds

Pneumomediastinum is discussed in Chapter 17 Wounds of the mediastinum should be treated as if infection had already occurred i e with drainage and antibiotics.

SUMMARY OF PRINCIPLES

1 Modern cardiovascular surgery is based upon an understanding of normal cardiorespiratory physiology and its pathologic derangement in disease In cardiovascular respiratory disease history and physical examination are still of paramount importance They are aided and abetted by new methods of examination The latter include new roentgenographic technics multiple lead electrocardiography cardiac catheterization, and angiocardiography

2 Technical modalities of therapy include the blind method, semiblind technics and surgical intervention under direct vision The latter method is attained by the use of hypothermia with inflow occlusion and preferably by extracorporeal circulation

3 Cardiac malformations occur before the end of the tenth week of embryonic life Although it is probable that certain virus diseases play a role in the pathogenesis of these malformations the genetics and chemistry of congenital defects are still obscure

4 Congenital heart defects may be in the cyanotic or acyanotic category Cyanosis may be due at least partially to a shunting of venous blood into the systemic arterial circulation through some type of defect or it may be due to the inability of blood from the right side of the heart to reach the lungs Dyspnea, particularly tachypnea is a regular companion to cyanosis Clubbing of the fingers and toes accompanies chronic cyanosis

5 Other cardinal signs in congenital heart disease in addition to cyanosis are murmurs enlargement of the heart, syncope polycythemia

pulmonary infections and pulmonary edema squatting stunting of growth increased fatiguability and congestive heart failure

6 The characteristic dynamic changes in a patent ductus arteriosus are those of an arteriovenous fistula and include a diminished diastolic pressure a *wide pulse pressure an increased circulating volume*, hypertrophy and ultimately dilatation of the left ventricle. Clinically the disease is manifested by underdevelopment, the characteristic to and fro machinery murmur of an A V fistula the "hilar dance on fluoroscopy, and the absence of cyanosis as long as the heart is competent Although not frequently necessary angiocardiography and cardiac catheterization may be definitive when the diagnosis is questionable Surgical therapy of patent ductus arteriosus is curative

7 Atrial septal defects are due to failure of closure of the septum between the two atria at various levels this defect may be found in association with other abnormalities Several methods of correction are available the most definitive being closure under direct vision

8 Most interventricular septal defects occur in the membranous portion near the left ventricular outflow tract. The shunt is usually from left *to right If this defect is found in association with congenital pulmonary* hypertension, the combination of these defects is known as the *Eisenmenger complex* Surgical closure of ventricular septal defects is most satisfactorily accomplished with the aid of extracorporeal circulation under direct vision

9 Pulmonic stenosis both valvular and infundibular interferes with the normal flow of blood from the right ventricle to the lungs and results in dyspnea fatiguability and cyanosis upon exertion Correction is accomplished either by the blind transventricular route or under direct vision through the open pulmonary artery

10 One of the most common congenital heart defects is the tetralogy of Fallot The pathologic findings are those of a high ventricular septal defect a pulmonic stenosis an overriding aorta and right ventricular hypertrophy In very young and very sick patients this complex anomaly can be alleviated by the shunting procedures which bring an arterial blood supply to the pulmonary circuit. When the patient can be more definitively prepared a *corrective procedure can be done by open heart technics* under direct *vision with the aid of cardiopulmonary bypass*

11 Aorticopulmonary window is an arteriovenous fistula with the same pathologic physiology as obtains in patent ductus arteriosus Because of the precarious position of this defect it can be corrected with safety only with the use of extracorporeal circulation technics

12 Congenital aortic stenosis is correctable by the same direct approach as acquired aortic stenosis

13 Tricuspid atresia is a severe cyanotic congenital *heart disease and* is compatible with life only if there is a shunt between the right and left atria Blood is supplied to the lungs via bronchial arteries and/or through a concomitant patent ductus arteriosus

14 Anomalous vascular rings of the aortic arch are usually diagnosed by pressure upon the trachea and/or esophagus Therapy is surgical and consists of division of the lesser ring Grafts may be used if necessary

15 Complete transposition of the great vessels implies the origin of the aorta from the right ventricle and of the pulmonary artery from the left ventricle. The best operation for the correction of this serious cyanotic disease to date is venous transposition the directing of pulmonary venous return from the right lung to the right atrium and anastomosis of the inferior vena cava to the left atrium

16 Rheumatic fever heart disease is the consequence of the healing process of an active endocarditis Granulation tissue progresses to cicatrization with resulting distortion of the valves and the chordae tendineae leading to corresponding impairment of function. The nature of the dynamic result depends upon the nature of the cicatrix and the valve involved

17 Mitral stenosis is one of the most common types of valvulitis of rheumatic fever heart disease Stenosis of the mitral valve results progressively in increased pressure within the left atrium which is then transmitted to the pulmonary veins Pulmonary hypertension and pulmonary vascular sclerosis ensue Pulmonary hypertension and right ventricular hypertrophy constitute the entity of chronic cor pulmonale Clinically dyspnea is due to pulmonary hypertension hemoptysis to rupture of the venous varices in the lung fatiguability and progressive reduction in exercise tolerance are due directly to diminished left ventricular output Mitral commissurotomy is the operation of choice in properly selected and prepared cases

18 Mitral regurgitation is due to the scarring of the valve in the open position or to loss of valve substance by curling of its edges or to dilatation of the mitral annulus Surgical therapy of mitral regurgitation is not yet as satisfactory as that of mitral stenosis however correction under direct vision by open heart technics are more promising

19 Aortic stenosis imposes an added work load upon the left ventricle in its attempt to eject a normal blood volume Myocardial ischemia due to partial occlusion of the coronary ostia may result in angina. Diminished left ventricular output may result in cerebral ischemia and syncope Left ventricular hypertrophy is the rule Correction of aortic stenosis is difficult and frequently unrewarding Replacement of this valve would be desirable

20 Aortic regurgitation leads to gigantic hypertrophy and ultimately to dilatation of the left ventricle No completely satisfactory operation for the correction of this disease has yet been devised The ball valve baffle operation in the descending aorta is palliative

21 One of the greatest scourges of our day is coronary artery heart disease It constitutes about 60 per cent of all heart disease seen clinically It is basically the process of atherosclerosis leading to obliterative arterial disease of the coronary arteries Ischemia may result in infarction or in diffuse fibrosis Infarction may lead to a mechanism death, the production of ventricular fibrillation by differences in electrical potential in two ad

jacent areas of muscle the late sequelae of myocardial infarction are embolic phenomena congestive heart failure and aneurysmal dilatation of the ventricular wall Medical therapy of acute and chronic coronary insufficiency is wanting Efforts toward the relief of chronic coronary in sufficiency by bringing an increased supply of blood to the myocardium may be directed in either of two pathways (*a*) bringing an extracoronary source of blood to the myocardium (*b*) the development of intercoronary anastomoses and therefore a better redistribution of blood available to the heart Clinically these operations in properly selected cases may protect against future occlusions reduce pain, and possibly increase longevity

22 Wounds of the heart may be inflicted by sharp instruments high velocity missiles or blunt trauma to the chest wall The latter must be suspected particularly in automotive and industrial accidents.

23 Circulatory arrest also known as *cardiac arrest* or *cardiorespiratory failure* is the term usually applied to the acute sudden cessation of an effective heartbeat *The heart may be in standstill, asystole or the ventricle* may be fibrillating In either case there is an arrest of cardiac and therefore of circulatory, function The cardinal factor in the causation of this ca tastrophe is hypoxia Vagal reflexes hypercapnia, pre-existent heart disease and reflexes initiated by traction on autonomic nervous system structures may be the precipitating and predisposing factors in the presence of hypoxia The treatment of this catastrophe falls clearly into two component parts (*a*) prophylactic (*b*) definitive The cardiac status of all patients should be carefully evaluated before they are subjected to surgical op•r ation. Hypovolemia due to blood loss must be avoided continuous adequate oxygenation under anesthesia must always be provided noxious reflexes may be abolished by adequate amounts of atropine in the preoperative and operative phases The definitive treatment includes positive pressure anes thesia with an indwelling endotracheal tube *immediate thoracotomy, and* the maintenance of circulation by manual systole If manual systole does not restore a beat to a heart which is in asystole epinephrine or calcium ion may be used to start a beat if ventricular fibrillation is present the heart may be defibrillated with a proper current An artificial pacemaker is very helpful The heart which is uncorrectably diseased is most difficult to resuscitate

24 The pericardium is a fibrous envelope which surrounds the heart and is attached to the origin of the great vessels and to the diaphragm It enlarges to accommodate gradual increases in size of the heart but it may constitute a limiting factor in acute dilatation of the heart

25 Pericardial effusions may be hemorrhagic serous or pyogenic in nature They may be acute or chronic the acute is usually traumatic and *the chronic usually due to metastatic malignant disease* Pericardial effu sions are seen in congestive heart failure and in inflammatory diseases the latter may be either tuberculosis or rheumatic fever The diagnosis of

pericardial effusion is made on the basis of the Beck triad which includes an elevated venous pressure a reduced systemic arterial pressure, and a small quiet heart on fluoroscopy Therapy includes aspiration of the fluid from the pericardium for relief of tamponade identification of the nature of the fluid and treatment of the underlying disease

26 Chronic constrictive pericarditis (the compressed heart) is the late sequel to conversion of fibrinous deposits and adhesions into mature fibrous tissue. This process obliterates the pericardial space and may lead to compression of the heart. The sequelae are inadequate diastolic filling of the heart and subsequently inadequate systolic ejection i e a diminution in stroke volume The inability of the left side of the heart to fill in diastole is productive of pulmonary hypertension The reduced left ventricular output results in severe dyspnea on exertion Compensatory mechanisms are tachycardia and elevated venous pressure Severe passive hyperemia results in hepatomegaly and ascites Clinically the disease is recognized by the Beck triad plus symptoms due to chronicity dyspnea and ascites, and electrocardiographic evidence of a specific nature Surgical therapy consists in pericardiectomy or cardiac decortication

27 Coarctation of the aorta is a congenital malformation in which there is a narrowing of the aortic lumen in the region of the ductus arteriosus All degrees of constriction occur In the infantile type the coarctation occurs proximal to a patent ductus arteriosus in the adult type the coarctation is found distal to the ductus arteriosus Collateral vascular dilatation around the coarctation is most marked in the adult type Clinically the disease is recognized by the high arterial pressure in the upper extremities with a low arterial pressure and peripheral ischemia in the lower extremities additionally the roentgenographic finding of huge intercostal vessels which may notch the inferior borders of the ribs, can be demonstrated. In the infantile type cyanosis and clubbing of the toes may be manifest. Cardiomegaly and congestive failure are common. Aortography may define the type and extent of the coarctation Surgical therapy of coarctation of the aorta is indicated in every case of the adult type, with or without a patent ductus arteriosus A graft may be used to bridge the defect if necessary

28 Arteriosclerotic aneurysms of the aorta are most common in the descending portion and its branches, particularly at their bifurcations They are treated by resection and replacement with homografts or appropriate prostheses Luetic aneurysms are more common in the ascending aorta, and recently even this segment has been successfully resected and replaced. Dissecting aneurysm is most common in the descending thoracic and abdominal aorta. The combination of hypertension and atherosclerosis usually is required to permit this set of circumstances. The dissection may continue to varying distances and may include the renal, mesenteric and common iliac arteries The De Bakey re-entry operation may be lifesaving

29 The mediastinum lies between the two pleuropulmonary spaces and

houses the thymus great vessels pericardium and heart esophagus and the root of the tracheobronchial tree in addition to the thoracic duct and vagus and sympathetic nerves

30 Mediastinitis may be due to chronic infection of lymph nodes, particularly by tuberculosis Direct extension from the pleuropulmonary spaces is not uncommon Acute infection of the mediastinal space may be due to instrumentation of the esophagus and of the tracheobronchial tree and to penetrating and perforating foreign bodies. The systemic signs of mediastinitis are those of a violent infection particularly if a phlegmonous mediastinitis exists Therapy follows the general principles of infection of a closed space anywhere in the body and includes drainage per mediastinotomy and supportive therapy together with the use of antibiotics

31 The common tumor of the anterior and superior mediastinum is the dermoid thymus tumors are rare, the most frequently found tumor in the posterior mediastinum is the neurofibroma arising from the intercostal nerves The most common tumors of the mediastinum are the lymphoma group in the hila of the pleuropulmonary spaces

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21

The Lip, Tongue, Mouth, Palate, and Pharynx

I. **Surgical anatomy and physiology**
II **Carcinoma of the lip**
III **Carcinoma of the tongue**
IV **Buccal mucosa and gingivae**
A Leukoplakia and cysts
B Epulis and tumors of dental origin
V **Pharynx and tonsils**
A Carcinoma
B Diseases of the tonsils
VI. **Salivary glands**
A Ranula
B Calculi

SURGICAL ANATOMY AND PHYSIOLOGY

The mouth is a strategic structure which to a very great extent, places the individual in contact with his environment It is the portal of entry of food and drink the chief means of communication by means of facial expression as well as of speech an adjunct to respiration, including intake and exhalation of air and a site for ejection of the products of more violent tussive efforts (coughing) to remove foreign matter from the bronchial tree. Additionally it is a zone of erogenous stimuli at all ages This area is, next to the skin, the chief barrier to the invasion of harmful microorganisms. For this reason an abundant blood supply and rich lymphatic drainage are essential The mouth and its associated structures are a part of the digestive apparatus and initiate the moistening and mastication of food by saliva the beginning of carbohydrate breakdown for this purpose specific muscles and secretions are required Cosmetically it dominates the facial features and its embellishment by the female is universal in most cultures For these multiple and varied purposes a large number of highly specialized organs and tissues are required. These are all involved in surgical problems affecting the mouth

CARCINOMA OF THE LIP

The lips are two fleshy folds which meet at the lateral commissures to circumscribe the buccal orifice The orbicularis oris muscle passes in the substance of the lips and aids in articulation essentially by closing the mouth It is attached to other facial muscles at the lateral commissures which cause the mouth to open. It is innervated together with the other muscles of facial expression, by the seventh cranial (facial) nerve The surfaces of the lip are skin on its exterior surface buccal mucosa on its inner surface and the vermilion border of the lip between the two

Cancer of the lip is the most frequent ma

lignant tumor in and about the mouth and occurs almost exclusively in the lower lip of males It is almost always an epidermoid (squamous) carcinoma As elsewhere the cause of cancer is unknown but both mechanical and actinic trauma are predisposing factors The diagnosis of cancer of the lip is made by the excision of any suspicious lesion and study of the histologic picture Early the lesion may look like herpes a wart an ulcer a weeping papule or a little white spot resembling leukoplakia. One never cuts into such a lesion but rather performs an excision biopsy without touching the lesion

Since this is an accessible lesion, the factor of greatest prognostic significance is not the size or position of the lesion but rather the extent to which lymphatic spread has occurred. The very abundant lymphatic drainage from the center of the lip is primarily to the submental nodes from the lateral commissures of the lips the spread may be to either the submental or directly to the submaxillary nodes From these two primary depots the lymph drainage then proceeds to the deeper cervical triangles and along the course of the internal jugular vein Although surgical extirpation and irradiation are both successful in a large percentage of cases, the former is to be preferred since the cosmetic results are better and there are no late deleterious effects It is the consensus that when a carcinoma of the lip is removed by a V-shaped excisional biopsy nothing further need be done at that time if no lymph nodes are felt in the submental or submaxillary areas or elsewhere in the neck. If lymph nodes are enlarged, they too must be excised for biopsy If they are involved by metastases a radical neck dissection should be performed. In general, the 5 year nonrecurrence rate is very high

CARCINOMA OF THE TONGUE

The tongue is a muscular organ which half fills the closed mouth cavity and has important functions in deglutition and phonation It is attached to the floor of the mouth to the mandible and to the hyoid bone It is covered by a heavy layer of squamous epithelium, and contains numerous specialized glands The blood supply to this structure is abundant the lymphatics are similarly extensive those of the anterior portion of the tongue draining into the submental and submaxillary nodes before going to the deep cervical chains and those from the base of the tongue draining directly into the deep cervical nodes The lymph channels decussate freely

Cancer of the tongue is common and occurs largely in males past middle age The most common sites of origin are at the lateral borders of the anterior two-thirds of the tongue Chronic irritation is thought to be a predisposing factor and includes such agents as tobacco leukoplakia, ill fitting artificial dentures and luetic infection.

The lesion is histologically almost invariably epidermoid carcinoma, and invasion into the rich network of lymphatics is almost always obvious when the tumor becomes symptomatic The deep lymph nodes of the neck are usually involved by the time the diagnosis is made A lymph node diagnosis may be made even before the lesion is discernible to the patient. Differential diagnosis includes luetic gumma, tuberculosis leukoplakia, aphthous ulcers benign tumors and chronic granulomas

It is worthwhile treating all areas of leukoplakia prophylactically since they are considered to be precancerous in character Attention should be directed early to chronic ulcers and to the replacement of ill-fitting dentures. Active treatment of cancer of the tongue is either by extensive surgical block excision of the tongue mandible and floor of the mouth and radical dissection of the neck (commando resection) or by various methods of irradiation. If cervical metastases occur after primary irradiation therapy they should be removed by radical neck dissection. Usually a combination of both methods is used at some stage in the over all unsatisfactory treatment of

this serious disease. The over all prognosis is not encouraging 30 per cent 5 year non-recurrence rate being the best reported by institutions using the most radical approach for the eradication of this malignant tumor

BUCCAL MUCOSA AND GINGIVAE

The mouth is lined throughout by squamous cell epithelium The gingivae are composed of a spongy substance covered by buccal mucosa. They clothe and protect the dentoalveolar junctions

Leukoplakia and Cysts

The most common lesions of the buccal mucosa are areas of leukoplakia and reten tion mucous cysts The lining is frequently subjected to trauma by repeated irritation from ill fitting dentures and not infre quently by external violence The blood supply and therefore the healing proper ties of the buccal mucosa are great. Most lacerations of the buccal mucosa need not be and preferably should not be closed by sutures Infection in such traumatic wounds is less frequent if they are left exposed to air rendering difficult the growth of anae robic microorganisms universally present in the mouth

Epulis and Tumors of Dental Origin

Epulis is a growth of the gingivoalveolar ridge which is considered by most patholo-gists to be a benign giant-cell fibrous tumor Others consider it to be an infectious granu loma The origin appears to be from dental periosteum Treatment is by complete ex cision of this histologically benign tumor Epulis and other lesions of the gingival mar gins must be differentiated from tumors and cysts of dental origin such as adamantino-mas (of enamel forming cell origin) dental root (dentigerous) cysts and tumors of bone

PHARYNX AND TONSILS

The pharynx is a musculomembranous vestibule behind the mouth below the nose and above the larynx Deviations from nor mal in this area are made manifest by dis-turbances in swallowing, breathing and phonation In normal respiration the oro-pharynx and nasopharynx are in continuity During deglutition the soft palate obliterates the nasopharynx, thus preventing ascent of food into the nasopharynx. The muscles of the pharynx are constrictors and elevators and are important in the normal process of swallowing. The retropharyngeal space is that loose areolar tissue between the poste rior pharyngeal wall and the prevertebral fascia which is continuous with the medias-tinum

Carcinoma

The nasopharynx as well as the hypo-pharynx (laryngopharynx) is not uncom-monly the site of squamous cell carcinomas. The eustachian tube drains the middle ear into the nasopharynx. Tumors of the phar ynx are frequently discovered for the first time through biopsy of metastatic lymph nodes in the neck.

Diseases of the Tonsils

The glossopalatine (anterior) fold and the pharyngopalatine (posterior) fold en-compass the palatine tonsil on each side The most frequently encountered diseases of this lymph structure are acute and chronic tonsillitis Treatment is by excision of the palatine tonsils preferably during a quiescent stage being careful not to injure the large blood vessels of the neck by tenting them into the operative field Peritonsillar abscess (quinsy) and retropharyngeal ab-scess have been discussed in Chapter 14 Squamous cell carcinoma and syphilis of the palatine tonsils are occasionally seen

SALIVARY GLANDS

Tumors and infections of the salivary glands have been discussed in Chapter 14

Ranula

Ranula is a cystic tumor of the floor of the mouth It is a result of obstruction of

22

The Esophagus

I Surgical anatomy and physiology; methods of study
II Anomalies
III Benign stricture
- *A* Accidental
- *B* Disease (1) esophagitis (2) scleroderma (3) web
- *C* External compression

IV Diverticula
- *A* Pharyngoesophageal
- *B* Epiphrenic

V Carcinoma
- *A* Diagnosis
- *B* Treatment

VI Hemorrhage
VII Perforations and rupture
VIII Dyskinesia
- *A* Achalasia (cardiospasm)
- *B* Dysfunction of esophagocardiac mechanism in hiatus hernia

SURGICAL ANATOMY AND PHYSIOLOGY, METHODS OF STUDY

The esophagus is a musculomembranous tube beginning proximally as a continuation of the pharynx at the level of the sixth cervical vertebra and extending distally to the esophagogastric junction at the level of the eleventh thoracic vertebral body It contains three areas of narrowing the first at its origin at the level of the cricoid cartilage the second at the level of the arch of the aorta (and bifurcation of the trachea) and the third at its termination It lies in the posterior mediastinum upon the prevertebral fascia, essentially in the midline being a trifle to the left at the level of its middle and third constrictions The esophagus is flattened in an anterior posterior direction in its empty state It is distensible and can be moved upward and downward as well as laterally In its cervical portion the esophagus is in intimate relation with the carotid sheath and its contained vessels on either side and with the recurrent laryngeal nerves which lie in the tracheoesophageal grooves The total length of the esophagus is approximately 25 cm in the human adult. An important measurement for purposes of examination is the distance between the incisor teeth and the esophagocardiac junction this approximates 40 cm in the adult. The esophagus passes through its hiatus in the diaphragm to reach its termination in the cardia of the stomach Thus, the terminal 6 cm of the esophagus is intraabdominal

The esophagus possesses no serosal coat. The musculature of the upper one third of the esophagus is striated in type In the middle third the fibers are mixed some being

striated others smooth In the distal third of the esophagus the muscle is entirely of the smooth variety The esophagus is lined with squamous epithelium Its blood supply is derived largely from direct branches of the aorta In addition the cervical esophagus receives some of its blood supply from the branches of the inferior thyroid artery and the lower esophagus is nourished by branches from the left gastric artery The lymphatic drainage of the esophagus is largely to the nodes of the mediastinum in intimate association with the tracheal carina the cervical esophagus is drained by nodes in the neck and the cardiac end of the esophagus by lymph nodes within the abdomen adjacent to the left gastric artery and the celiac axis.

The esophagus conducts food from the pharynx into the stomach Food and saliva are transmitted into the esophagus by the act of deglutition then propelled downward by esophageal peristalsis to the stomach The esophagus is innervated by efferent branches of the vagus nerves. The strength of the esophageal peristaltic waves depends upon the nature of the material being conducted as is true of any muscular tube and upon the size of the food bolus. Obstruction to the lumen of the tube increases the severity of peristaltic contractions in a downward direction until, ultimately reverse peristalsis occurs which results in regurgitation.

Dysphagia impaired swallowing, is due either to a disturbance in the complex neurologic mechanism of deglutition or to obstruction of the esophageal lumen by intrinsic occlusion or extrinsic pressure The esophagus is studied most effectively by two main methods First, roentgenography with the use of a thick radiopaque material is helpful in demonstrating disturbances of both anatomy and physiology The interior of the esophagus can be examined by *esophagoscopy* which also permits the taking of biopsy specimens.

ANOMALIES

The anomalies of the esophagus, seen early in life are discussed in detail in Chapter 23 These defects consist essentially of stenoses atresias tracheoesophageal fistulas, and webs

BENIGN STRICTURE

Accidental

Stricture of the esophagus from the ingestion of caustic chemicals is a very frequent occurrence particularly in children who swallow lye or other household corrosives. These materials are usually of such concentration as to cause instantaneous full thickness burns of the mucosa. They are followed by varying degrees of cicatrization which may be segmental in nature or diffuse and total and which may convert the esophagus into a fibrous cord. Early vigorous treatment is essential to prevent permanent occlusion and consists of the immediate administration of antidotal substances At the same time the patient is made to swallow a weighted string to facilitate later treatment. Pain shock, and fluid and electrolyte disturbances must be controlled as in any severe burn. A gastrostomy may be necessary for feeding

After the acute manifestations have subsided attention is directed toward maintaining the patency of the esophageal lumen. Repeated bougienage with graded sizes of dilators, guided by means of the string previously placed, may be necessary at frequent intervals for a long time in order to minimize the cicatricial stenosis If this is unsuccessful, replacement of the esophagus may become necessary either by bringing the stomach into the chest above the level of the stricture or by replacing the esophagus with an appropriate segment of the intestinal tract.

Disease

Stricture of the esophagus may also be caused by inflammatory diseases secondary to the presence of ectopic gastric mucosa or diverticula near the lower end of the gullet Commonly *peptic esophagitis* is due to re



FIG. 36. Stricture of esophagus due to severe peptic esophagitis. *A* Lateral view *B* posteroanterior view Treated successfully by resection and high esophagogastrostomy

gurgitation of highly acid gastric contents in association with peptic disease of the duodenum plus dysfunction either at the pyloric or cardiac ends of the stomach or at both (Fig 36) Cicatricial or spastic pyloric stenosis secondary to duodenal ulcer creates both these factors At the cardiac end, hiatal hernias interfere with sphincteric function and permit acid reflux Prolonged use of nasogastric tubes introduces direct trauma as well as prevents closure of the esophagogastric junction Management of the esophagitis is directed principally toward eliminating the primary cause Every effort is made to reduce an elevated acid gastric secretion This may not infrequently involve the surgical therapy of associated peptic disease of the duodenum. At any rate, it usually requires provision of a more adequate outlet for acid gastric contents from the stomach distally such as is afforded by pyloroplasty Repair of a hiatus hernia, with restitution of the normal esophagocardiac angle may be curative Recurrent ulceration and healing, often repeated will result in a cicatricial stenosis in the esophagus just as it does in the duodenum In such instances resectional therapy is indicated

Scleroderma a rather uncommon generalized disease of collagen tissue frequently involves the esophagus It is a disease of unknown cause in which there is hardening of many tissues, particularly the skin, capsules of joints, and sometimes the esophagus, with loss of its normal distensibility Dysphagia is usually the presenting symptom when the esophagus is involved. Therapy is directed at the generalized disease

Benign strictures due to esophageal intraluminal webs are occasionally seen in adult life without previous manifestations of esophagitis Their cause is unknown, but they are thought to be of congenital origin. The treatment is surgical correction.

External Compression

Obstructive stenosis from external pressure by enlarged mediastinal lymph nodes is occasionally seen Other causes of extrinsic compression of the esophagus are aortic aneurysm in adult life and anomalous vascular rings in the newborn infant.

DIVERTICULA

Diverticula are found in the esophagus as they are in other portions of the alimentary tract The usual classification is that of *pulsion* or *true diverticulum* and *traction* or *false type* The pulsion type consists of an outpouching of mucosa and submucosa through a weakened area of the esophageal wall. The traction type of diverticulum is not truly a diverticulum but is an outpouching of the entire wall due to pull from without by inflammatory adhesions The clinical manifestation of the traction type is usually

pain upon swallowing, or manifestations may be completely lacking. Occasionally symptoms may be due to an esophageal fistula. The *lymphadenopathy* which is responsible for this disease is usually secondary to tuberculosis of the hilar lymph nodes. Treatment is directed at the tuberculosis.

Pharyngoesophageal

True *pulsion diverticula* occur at the extremities of the esophagus. The most commonly encountered arises from the posterior pharyngeal wall above the cricopharyngeus muscle and usually presents to the left side of the neck in the posterior carotid triangle. The diagnosis is suggested by the history of regurgitation of food ingested many hours or even days previously, borborygmi in the neck, some degree of dysphagia, and foul breath. It is confirmed by roentgenographic demonstration of a blind pouch in the neck which becomes filled with ingested radiopaque material. Excision of the diverticulum is curative.

Epiphrenic

Epiphrenic pulsion diverticulum of the esophagus is less commonly encountered and is treated in much the same way. Diagnosis is made on the basis of roentgenographic demonstration of radiopaque material in a blind pouch just above the diaphragm, also most commonly to the left, associated with a train of events not dissimilar to that which occurs in the higher lying diverticulum. Additionally, substernal pain may be experienced. Peptic ulcer within the diverticulum, due to retaining acid secretions for long periods of time, is not infrequently seen in this variety.

CARCINOMA

Cancer of the esophagus is almost exclusively of the epidermoid (squamous) type. Adenocarcinoma is occasionally seen at the lower end, but in all probability these carcinomas really arise in gastric mucosa. There is only an occasional tumor of mesodermal origin (sarcoma) beginning in muscle or lymphoid structures. Although carcinoma of the esophagus may occur at any level throughout its length, the most common is in the midesophagus. Lesions of the lower end of the esophagus and of the cardia of the stomach may take their origin from either viscus.

Diagnosis

The diagnosis of cancer of the esophagus is made on the basis of its typical history of dysphagia and pain and on the roentgenographic demonstration of the tumor. The dysphagia is characteristically insidious in onset and is noted at first only with solid foods and finally even with liquids. Pain is a late symptom and may be due to involvement of nerves in the mediastinum or may result from tertiary peristaltic waves attempting to pass a bolus through the luminal obstruction. The diagnosis is further confirmed by exfoliative cytology and by endoscopic biopsy. Unfortunately, the diagnosis is seldom made early enough to permit a cure, because the vital structures of the mediastinum (including the trachea, bronchi, pericardium, and great vessels) are usually involved by the time exploration is undertaken.

Treatment

Treatment of cancer of the esophagus, as of cancer elsewhere, is wide resection and some type of reconstruction. The operative procedures vary with the site of the neoplasm. Even in the neck where conditions are more favorable for resection and reconstruction, the presence of regional lymph node metastases usually precludes complete cure. The region which is most amenable to earlier diagnosis and resection is the lower end of the esophagus, although here, too, subphrenic, paragastric, and hepatic metastases may already exist at the time of exploration. Cancers of the midportion of the esophagus have almost always invaded adjacent structures in the mediastinum (trachea, bronchi, perivascular sheaths, and pre

vertebral fascia) at the time of initial operation. Even though resection and restoration of continuity are possible prolonged survival is precluded by the aforementioned extension of the disease to nonresectable areas In line with tendency toward increasingly wide excision in the surgical management of malignant disease resections of the esophagus have become more radical in recent years with replacement of the gullet by portions of the large or small bowel interpolated between the pharynx and the stomach. Other means of replacement may be by prostheses (autologous skin or inanimate objects) or by elevation of the stomach Palliation in inoperable situations may consist of gastrostomy for feeding purposes or the insertion of an intraluminal tube to maintain patency

HEMORRHAGE

Hemorrhage from the esophagus is one of the frequent causes of massive upper gastrointestinal bleeding It is almost always due to bleeding from esophageal venous varices secondary to portal hypertension The diagnosis of bleeding from varices of the esophagus may be extremely difficult, particularly in view of the emergency situation which massive hemorrhage entails The patient usually gives a history which is compatible with the diagnosis of portal hypertension and may have associated suggestive findings, such as dilated veins in the abdominal wall and hepatomegaly The demonstration that the blood originates from the esophagus and not from the stomach may be made endoscopically or by use of the multiple balloon (Sengstaken) tube Roentgenographic demonstration of greatly dilated veins in the esophageal submucosa is often possible and is confirmatory

Severe esophageal bleeding is a dire emergency and its treatment taxes the skill and ingenuity of those responsible A logically planned attack must proceed in sequence from the simplest to the most extreme measure as circumstances dictate This begins with replacement therapy to overcome the effects of hemorrhage and shock, and which may alone suffice If bleeding continues, the use of an intraesophageal balloon to compress the submucosal veins is an effective temporary measure Emergency open operation is reserved for otherwise uncontrollable cases Transthoracic transesophageal obliteration by suture of the dilated, thin-walled vessels may be done at the time or may follow successful nonsurgical emergency management. Endoscopic obliteration of these veins by injection of sclerosing materials is seldom successful

Although treatment of massive bleeding from varices of the esophagus is urgent and lifesaving attention must later logically be directed toward the treatment of the primary underlying disease Definitive management of portal hypertension is discussed in a subsequent chapter The concomitant treatment of associated peptic esophagitis merits emphasis since many surgeons believe that inflammation is a necessary prerequisite for the erosion of submucosal veins

PERFORATIONS AND RUPTURE

Any break in the continuity of the esophageal wall is extremely hazardous. The escape of infective material into the areolar tissue of the mediastinum leads to a rapidly spreading cellulitis The esophageal wall may be breached by perforation or by rupture Perforation occurs as a result of penetrating injuries, in which there is usually serious associated damage to contiguous important structures Ingested sharp-pointed foreign bodies and endoscopic manipulation may also result in penetration Ulcerating lesions such as peptic ulcers or carcinomas, may also perforate the esophageal wall Rupture of the esophagus is occasionally seen as a result of increased hydrostatic pressure and accompanies severe and protracted vomiting This most commonly occurs in the epiphrenic region on the left side

The pathology secondary to perforation or rupture of the esophagus is essentially a

diffuse phlegmonous cellulitis of the mediastinum. It may be accompanied by pleuritis if the mediastinal pleura is breached. The diagnosis is made on the symptoms of acute substernal pain usually radiating to the left chest and frequently associated with dyspnea in varying degree and shock. Roentgenographic examination will usually disclose the presence of air in the mediastinum and fluid in the left chest. Colored solutions given by mouth may be recovered from the left pleural space. Systemic findings include a rapidly rising fever, leukocytosis and the toxicity associated with serious diffuse infections.

All esophageal perforations, penetrations, or ruptures demand immediate and emergent treatment. Momentary pin point perforations may become sealed off and require no operative intervention. Occasionally *simple mediastinotomy* will suffice to permit drainage of the infection, while the breach in the wall closes spontaneously. If there is associated pleural contamination drainage of the pleural space associated with esophageal aspiration through a nasogastric tube may allow a small hole to be sealed off. Finally open thoracotomy with *suture of the rent* is necessary in gross ruptures of the wall. Treatment of the underlying associated disease is always indicated.

DYSKINESIA

Achalasia (Cardiospasm)

Achalasia is a functional stenosis of the terminal end of the esophagus with proximal dilatation and associated dysphagia and vomiting. The esophageal distention may reach huge proportions with progressively more severe symptoms.

The cause of achalasia is unknown, but because of its similarity to hypertrophic pyloric stenosis and congenital megacolon a neurologic dysfunction is assumed. Milder forms may be treated by diet, bougienage, and antispasmodics. Severe cases require surgical therapy. The operation of choice is esophagocardiomyotomy (Heller procedure) which is similar in principle to the Fredet Ramstedt operation for pyloric stenosis and which affords a great measure of relief. The older methods of resection or esophagogastrostomy frequently lead to regurgitation esophagitis and have been largely superseded by the Heller operation.

Dysfunction of the Esophagocardiac Mechanism in Hiatus Hernia

The junction of the conduitlike esophagus with the cardia of the stomach is not just a simple transition from a narrow tube to a ballooned-out receptacle. Nor is it an ordinary sphincter. Rather, it is an involved mechanism associated with complex muscular movements, which are incompletely understood. It is well described as the *esophagocardiac mechanism*. The esophagus enters the cardiac end of the stomach obliquely, the right wall of the esophagus being continued as the lesser curvature of the stomach in what is almost a straight line. The left wall of the esophagus joins the cardia at an acute angle. Cinefluorophotography reveals that in the process of transmission the bolus of food is ejected from the esophagus into the stomach. This action is followed by a minimal regurgitation of gastric contents into the distal 1 or 2 in. of the esophagus. At that moment, a complex action of esophageal and gastric musculatures, together with diaphragmatic contraction, prevents further reflux. This action is dependent upon the acute left esophagogastric angle described above. When this relationship is disturbed, regurgitation of acid gastric contents into the esophagus occurs with resultant esophagitis. The symptom most commonly associated with this disturbance is *pyrosis* (*heartburn*). Gastric hyperacidity and pyloric obstruction particularly predispose to acid regurgitation and esophagitis. Additionally esophagogastric dyskinesia occurs when there are anatomic derangements in this area. The most common of these are diaphragmatic hernias of the esophageal hiatus types.

Congenital short esophagus is occasionally referred to as a *congenital hiatus hernia* In this situation the esophagus is so short that it ends in the thorax instead of below the diaphragm and the proximal portion of the stomach lies within the mediastinum The esophagus joins the stomach in a straight end-to-end fashion without the acute angle relationship which is normally present on its greater curvature side This anomaly strictly speaking is not an esophageal hiatus hernia but represents a disturbed anatomic and later functional, relationship between the two structures involved

Acquired esophageal hiatus hernias are of two main types In the *paraesophageal* type the normal esophagogastric junction remains within the abdomen, but a portion of the stomach is herniated through the hiatus to one side of the esophagus usually the left The distortion of the greater curvature of the stomach under these conditions disturbs the normal angle between the greater curvature and the esophagus. Furthermore, acid gastric contents are retained in the trapped portion of the stomach that lies within the chest. The high acid content of this sac not infrequently produces a peptic ulcer with its usual sequelae A *sliding esophageal hiatus hernia* is one in which the wall of the stomach and the esophagus are pushed up into the mediastinum, elevating the esophagogastric junction above the diaphragm When this anatomic situation obtains there is complete distortion of the junction of the esophagus and stomach with resultant *dyskinesia* This type of esophageal hiatus hernia is sometimes referred to as the *acquired short esophagus* It is not a short esophagus at all it is a displaced one This is the most common variety of hiatus hernia (Fig. 37)

Closure of esophageal hiatus hernia usually does not relieve the symptoms unless

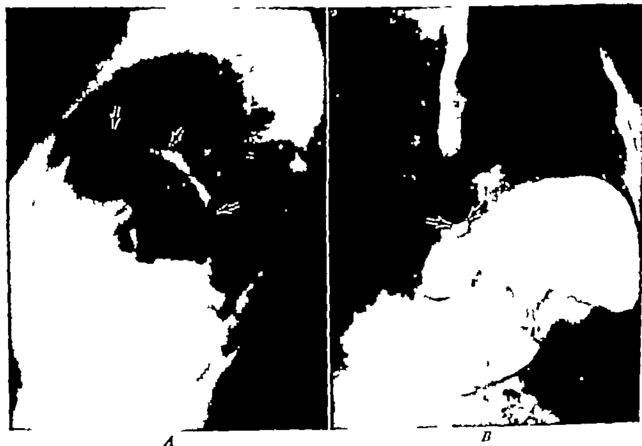


FIG 37 *A* Left lateral view showing stomach in the left chest. *B* Anterior view following ingestion of barium showing the stomach in the chest and outlining an epiphrenic diverticulum of the esophagus

the normal anatomic relationship between the esophagus and the stomach is restored This is essential in order to prevent re gurgitation of gastric contents into the esophagus This is understandable since it is the disturbed physiology of the area rather than merely the anatomic derangement which is productive of symptoms This may be expressed in another way by stating that the indications for repair of a hiatus hernia are really the presence of complications that it produces These complications are (1) the production of respiratory distress by encroachment on the lung (2) irritation of the pericardium with pain that cannot be differentiated from coronary artery heart disease and (3) the occurrence of peptic disease within the herniated stomach or in the esophagus

SUMMARY OF PRINCIPLES

1 The esophagus is a specialized conduit carrying food from the pharynx to the esophagus, whose function is not simple at all It receives food from the pharynx by a complex process of deglutition moves the bolus along by an intricate system of peristalsis and discharges the food into the stomach through a complex neuromuscular arrangement with the stomach the cardioesophageal mechanism

2. The esophagus contains no serosa lies largely within the medias tinum and is in intimate relationship with the vital structures of the respiratory and cardiovascular systems

3 Dysphagia may be due to a disturbance in the neurologic mechanism of deglutition or to obstruction of the esophageal lumen from within or without.

4 Anomalies of the esophagus are usually detected very early after birth, and are discussed in another chapter

5 Benign strictures of the esophagus are usually due to the ingestion of corrosive chemicals by children Early treatment is the treatment of shock and vigorous attempts to avoid cicatricial stenosis of the esophagus The latter involves the swallowing as early as possible, of a weighted string with the weighted object in the stomach

6 Graded bougienage should be tried for dilatation of the esophagus before reconstructive esophageal operations are attempted The latter may be accomplished by restitution of continuity using autologous tissues (skin or intestinal tract) or prostheses

7 Peptic esophagitis is the most common disease which leads to benign stricture of the esophagus Treatment is directed toward removal of the underlying disease, which is frequently peptic disease of the duode num and reconstructive measures of the esophagus Pyloroplasty is usually necessary

8 True diverticula of the esophagus are pulsion in nature. They oc cur at the extremities of the esophagus, most commonly in the cervical esophagus through the cricopharyngeus muscle epiphrenic diverticula occur just above the diaphragm, usually to the left side Pulsion diverticula consist of mucosa and submucosa

9 False diverticula are due to traction from without by inflammatory adhesions usually due to tuberculous lymph nodes they consist of the entire esophageal wall

10 Cancer of the esophagus is almost exclusively squamous in type The symptoms are insidious in character and consist essentially of dysphagia and pain and the roentgenographic demonstration of a tumor

11 Except for cancer of the lower portion of the esophagus the diagnosis is rarely made in time to permit successful resection and longevity In the midesophagus, vital structures of the mediastinum are usually invaded early in the course of the disease

12 Hemorrhage from the esophagus may threaten life and is usually due to esophageal varices secondary to portal venous obstruction Esophageal hemorrhage is treated by correction of shock and tamponade of the bleeding veins. Emergency suture of the veins may be necessary The underlying disease is treated after recovery from the hemorrhage

13 Perforations and rupture of the esophagus result in a rapidly spreading phlegmonous mediastinitis Perforations are caused either by trauma or by disease Rupture of the esophagus is due to raised hydrostatic pressure in the esophagus as in severe and protracted vomiting Therapy of perforated or ruptured esophagus may require mediastinotomy for drainage of the mediastinum and operative closure of the gullet.

14 Dyskinesia of the esophagocardiac sphincter mechanism is usually due to destruction of the normal anatomic relationships in this area, followed by reflux esophagitis Hiatus hernias of various types may lead to such dyskinesia The management consists of reduction of the hernia and reconstruction of the normal relationship between the esophagus stomach and diaphragm

15 Achalasia of the esophagus is a disease similar to hypertrophic pyloric stenosis and congenital megacolon the cause of which is probably a neurologic dysfunction Correction is by esophagocardiomyotomy

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23

Anomalies and Malformations of the Alimentary System

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- *B* Type of disease produced

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X Diaphragmatic anomalies: hernia and eventration

XI Colon rectum, and anus
- *A* Atresia and stenosis
- *B* Imperforate anus and fistulae with genitourinary tract
- *C* Hirschsprung's disease (congenital megacolon)

BASIC EMBRYOLOGIC CONSIDERATIONS

Definitions

The alimentary system is formed early in embryonic life from an invagination of endoderm into a primitive tube Originally a solid cord of cells a lumen appears at either end at about the fourth week con stituting the *stomatodeum* at the cephalic end and the *proctodeum* at the caudal end By the fifth week of fetal life the intestinal tube has developed a lumen throughout which is lined with epithelium During the next 6 weeks this lumen becomes obliterated by epithelial debris At about the twelfth week this solid gut again becomes vacu olized and patency is re-established Any arrest in the re-establishment of the lumen at this time results in *atresia* (literally no hole) causing a discontinuity of the intesti nal canal. *Stenosis* or narrowing of the lumen, may be due to incomplete re-establishment of the gastrointestinal lumen it may involve a considerable length of gut, or it may be merely a thin septum across the bowel which is perforated by a small aperture Atresias and stenoses similarly may occur wherever a tubular structure is formed. In the alimentary tract they are most commonly found in the esophagus in the duodenum in the terminal ileum, and at the anus Not infrequently the areas of arrested vacuolization are multiple and result in several areas of occlusion in the same subject The mechanism responsible for them is unknown.

Type of Disease Produced

The genetics of congenital defects are poorly understood although certain virus diseases in the mother between the fifth and twelfth week of gestation may be contribu tory In general, the clinical effect of con genital malformations of the alimentary system is intestinal obstruction The symptoms, diagnosis and therapy of occlusion of each segment of the gut will be taken up under separate headings.

MOUTH, PALATE, AND PHARYNX

Cleft Lip and Cleft Palate

The lips buccal cavity and upper respira tory passages are formed during the sixth to eighth weeks of fetal life from the original stomatodeum Failure along the usually smooth course of events in this area results in the formation of the commonly known defects of *cleft lip* (harelip) and *cleft palate* These lesions may be of varying severity from a furrow in the upper lip to complete absence of the palate with communication between the buccal cavity and the paranasal sinuses

These defects of course are almost always detected immediately after birth. Cor rection of these lesions is timed according to the extensiveness of repair required and with due consideration to the psychic trauma that follows so frequently in children if lesions are not adequately repaired.

Branchial remnants in the neck ears, floor of the mouth and pharynx are discussed in detail in Chapter 14

ESOPHAGUS

Stenosis and Web

Malformations of the esophagus are of several types Stenosis, without atresia of the lumen is usually seen in the midportion of the esophagus. Difficulty is not experienced as a rule until solid food is ingested. A web is occasionally present with a small aperture in its center most frequently at the lower end of the esophagus. Atresia of the esophagus is a much more serious anomaly Not infrequently it is associated with a tracheoesophageal fistula, which further increases the gravity of the malformation. The accompanying diagram illustrates the vari ations in these lesions Type *C* is by far the most common, comprising about 90 per cent of all these anomalies (Fig 38)

Atresia and Tracheoesophageal Fistula

Atresia of the esophagus should be diagnosed in the first few days of life Tra

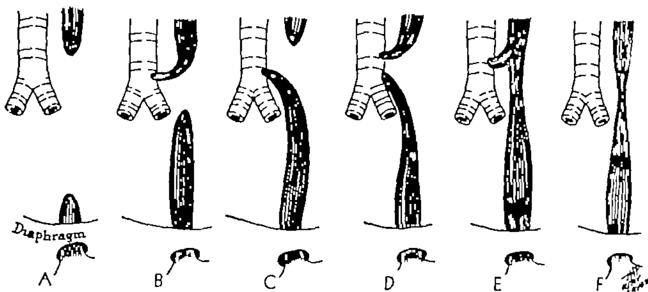


FIG 38 Congenital anomalies of the esophagus, including tracheoesophageal fistulae *A* Esophageal atresia no fistula. *B* Esophageal atresia proximal tracheoesophageal fistula. *C* Esophageal atresia distal tracheoesophageal fistula at or just above the carina This type comprises 90 per cent of all esophageal abnormalities. *D* Esophageal abnormalities. *E* Tracheoesophageal fistula no atresia *F* Esophageal stenosis.

cheoesophageal fistula associated with the atresia *must* be diagnosed early The outstanding symptoms and signs are (1) *excessive salivation* due to the inability of the oral secretions to pass down the esophagus (2) *respiratory distress* from inhalation of esophageal contents into the bronchial tree (3) *regurgitation* and (4) *coughing* especially at the time of feeding In premature infants esophageal atresia is the most common of all congenital anomalies Associated anomalies frequently occur in the distal end of the gut and in the heart.

Diagnosis of atresia can be made when a soft catheter cannot be passed down the esophagus and into the stomach If any doubt exists, a very small amount of radiopaque fluid may be instilled into the proximal esophagus and the blind end visualized radiographically An associated tracheal fistula frequently will be outlined Roentgenographic absence of air in the stomach almost always indicates an atresia of the esophagus with a communication with the trachea Presence of air in the stomach and the inability to pass a catheter down the esophagus make the diagnosis of esophageal atresia with a communication between the lower end of the esophagus and the tracheobronchial tree Because of regurgitation, *aspiration pneumonitis* is common in these babies

Simple stenoses and webs with relatively large apertures may be treated by repeated gentle dilatation with appropriate instruments Severe stenoses and all forms of atresias must be submitted to operation The first successful reconstructions of esophageal atresias were reported simultaneously by Ladd and Leven in 1939 In essence both writers re-established continuity by forming a subcutaneous tube from a segment of small intestine The method of choice today when feasible is that described by Haight two years later namely an intrathoracic primary repair The fistula is first servered and the tracheal wall closed Reconstruction of the esophagus follows The latter is usually accomplished by an end-to-end anastomosis of proximal and distal segments If the distal end is too short it may be necessary to perform an esophagogastrostomy The preoperative preparation of these tiny patients with this serious defect is most important Dehydration inanition, and often aspiration pneumonitis can be combatted within 24 hr Postoperative care must be similarly meticulous. Correction of this le

sion is one of the triumphs of modern surgery. Attention must be paid simultaneously to associated congenital malformations.

STOMACH

Hypertrophic Pyloric Stenosis

Hypertrophic pyloric stenosis is a relatively common congenital condition in which the signs and symptoms of obstruction to the outlet of the stomach become manifest 2 to 6 weeks after birth. The cause of the disease is not clear. There is, characteristically a striking hypertrophy of the musculature of the pylorus producing a smooth movable olive-shaped tumor which occludes the gastric outlet. Although numerous explanations for the occurrence of this pyloric muscular tumor have been advanced including that of aganglionosis (similar to Hirschsprung's disease) none are completely satisfying. This anomaly occurs principally in male infants, with an apparent predisposition in the first born.

Symptomatology consists mainly of vomiting which usually starts about the second week of life. Initially the vomiting is merely postcibal regurgitation, but later it becomes forceful. The vomitus does not contain bile. The baby seems to be hungry all the time and usually there is an associated weight loss. If neglected, obvious dehydration is seen and alkalosis is common.

Physical examination may reveal dilatation of the stomach particularly after feeding. Visible peristalsis may be observed, this being the extreme attempt of the stomach to force its contents through the obstruction. In the largest percentage of the cases the olive-shaped mass can be felt in the right upper quadrant beneath the liver edge. The diagnosis is further confirmed by x-ray examination, which shows an almost total gastric retention. Occasionally a fine line of barium can be seen to pass through the markedly narrowed lumen within the hypertrophied muscle (Fig. 39). Such a

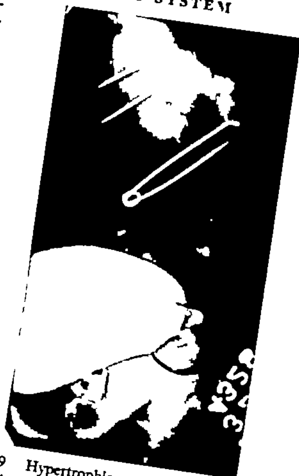


FIG. 39 Hypertrophic pyloric stenosis. Notice the retention of the barium in the stomach, with only a trickle of barium going through pylorus.

sign, with gastric retention and the history of vomiting without bile is diagnostic.

Other

Other conditions which cause vomiting, weight loss, and dehydration in the neonatal period are pylorospasm, atresia of the esophagus and obstructions of the small bowel. Spastic occlusions of the pylorus are usually intermittent, have no palpable tumor and respond to dietary and medical therapy. Atresia of the esophagus causes vomiting from the very first feeding, in addition to spillage of saliva and respiratory difficulties. Obstruction at a level below the entrance of the common duct into the duodenum will be associated with vomitus which contains bile. Head injury due to birth trauma must, of course be suspected in neonatal vomiting.

The treatment of hypertrophic pyloric stenosis is *pyloromyotomy* as described in-

dependently by Fredet and Rammstedt, the former in 1908 and the latter in 1911 This operative intervention is never an emergency There is always sufficient time to restore adequate hydration and nutrition by the parenteral route The operation consists in dividing the thickened pyloric muscle *longitudinally* until the mucosa pouts through Since the lumen of the stomach is not entered, feedings may be started soon after the operation In the postoperative care meticulous attention to nutrition and to fluid and electrolyte balance is mandatory Results are uniformly good

DUODENUM

Atresia, Stenosis, and Web

Of all congenital obstructions of the small intestine atresias and stenoses of the duodenum are second only to those of the terminal ileum Vomiting occurs shortly after birth and the stomach is markedly dilated after feeding The presence or absence of bile in the vomitus will be determined by the location of the site of obstruction with reference to the entrance of the common bile duct. Dehydration and inanition progress rapidly unless surgical therapy is carried out early In all complete atresias of the gastrointestinal tract no cornified cells can be found in the meconium (negative Farber sign) Such cells are found in the normal meconium and are due to swallowed vernix caseosa in intrauterine life Roentgenographic examination when an atresia of the distal duodenum is present, will reveal a duodenum almost as large as the stomach with an abrupt termination of the gas pattern Gas is absent in the small and large bowel distal to obstructions

Surgical therapy must be preceded by decompression of the hugely dilated stomach and duodenum Operation is fraught with danger because anastomosis is difficult In general the collapsed bowel distal to the atresia is so small that a side to-side reconstruction must be performed in these babies One should look for other associated and concomitant atresias even after one is found indeed *because* one is found In general, in these infants gastroenterostomy should be avoided and duodenojejunostomy performed. This is to avoid the churning back and forth of ingested food in the blind loop of the duodenum

Obstruction by Tight Superior Mesenteric Artery

Obstruction of the duodenum in the neonatal period may also be caused by compression from short superior mesenteric vessels and by external bands passing over the duodenum anteriorly toward the right parietal peritoneum The latter are more appropriately discussed under the heading of Malrotation of the Gut.

Obstruction by Annular Pancreas

A relatively uncommon reason for obstruction of the duodenum is its external compression by the pancreas growing circumferentially around it the annular pancreas The degree of obstruction varies greatly from complete, in which case the obstruction is usually detected in the neonatal period to relatively minor under which conditions symptoms may be minimal and appear later in life If the constriction is severe enough to produce symptoms, roentgenographic examination usually reveals a partial or relatively complete obstruction of the second portion of the duodenum

Treatment of choice in all duodenal obstructions is a duodenojejunostomy in the very young and gastroenterostomy in the older child or adult. As with other symptomatic congenital malformations associated lesions should be corrected at the same time if at all possible

JEJUNUM AND ILEUM

Atresia and Stenosis

Atresias and stenoses in the midgut are similar to those in the esophagus and duo-

denum Actually the most common site of atresia of the small intestine is in the terminal ileum near the ileocecal junction it occurs twice as often as atresia of the duodenum Stenosis, however, is more frequently found in the duodenum than in the ileum Both lesions are less common in the jejunum than in either of the previously mentioned portions of small intestine Obstructive lesions of the terminal ileum are associated with greater abdominal distention than those of the upper reaches of the gastrointestinal tract. They are not detected as early and the patients are usually in severe water and electrolyte imbalance when first seen by the surgeon Therapy consists of surgical correction of the obstruction as soon as dehydration and electrolyte imbalance are corrected

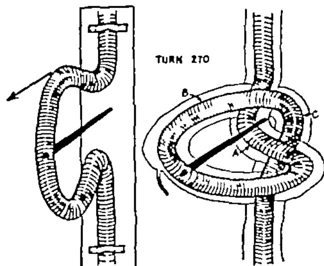


FIG. 40 Model showing rotation of the intestinal tube around the axis of the superior mesenteric artery in a counterclockwise fashion *A* Future jejunum. *B* Future ascending and transverse (right) colon. *C* descending (left) colon. (*Modified from Chaffin and Snyder*)

Malrotation of the Gut

Malrotation of the intestinal tract can present a most baffling set of circumstances on the operating table and give the surgeon a most gratifying feeling when he is able successfully to correct this anomalous defect.

Embryologic rotation and fixation of that portion of the intestinal tract which derives its blood supply from the superior mesenteric artery (the midgut) is a fascinating sequence of events that occurs early in embryonic life During the sixth to the tenth weeks of fetal life a portion of the midgut protrudes from the coelomic cavity into the umbilical cord. At the end of that time the coelomic cavity has grown to accommodate this portion of the alimentary tract, which then is returned to its normal habitat. During this process the proximal portions of intestine return first. As the gut returns, it rotates upon its common mesentery in a *counterclockwise direction* (Fig. 40) The junction of the small and large intestine (ileocecal area) initially lies in the left upper quadrant of the abdomen. From this position it migrates transversely to the right upper quadrant and then descends to its eventual position in the right lower quadrant. The left colon becomes fixed against the posterior abdominal wall on the left side just as the ascending colon is attached to the right The small intestine is similarly rotated in its inframesocolic position. Arrest of this rotational process at any point short of completion constitutes a malrotation A large variety of anomalies may occur If there is moderate delay in the return of the small bowel to the coelomic cavity the entire small intestine or some portion of it may become trapped behind the mesentery of the right colon a condition commonly referred to as *right paraduodenal hernia.* Greater delay permits inclusion of the small intestine behind the mesentery of the left colon, a *left paraduodenal hernia.* More frequently the cecum swings free on a long mesentery which has not become attached to the right parietal peritoneum

Many malrotations produce no symptoms. Some persons have lived to full life expectancy without serious symptoms Occasionally a very primitive arrangement of a *mesenterium commune* is seen in which the entire intestinal tract from the duodenum to the rectum is suspended by the dorsal mesentery which is attached to the posterior

midline More commonly however, the derangements which result from malrotation become manifest early in life This consists of *high intestinal obstruction* most commonly of the third portion of the duodenum by bands folds or a volvulus of a part or all of the midgut. The predisposition to *volvulus* is accounted for by the fact that the root of the mesentery is extremely narrow being only 1 or 1½ in in width Such a situation is conducive to twisting of the entire fanlike arrangement of the intestinal coils Twisting of the small intestine is usually in a clockwise direction and may be of 180°, 360° or even 720° This type of volvulus produces intestinal obstruction by two mechanisms (1) obstruction of the gastrointestinal lumen at one or more points (2) strangulation due to torsion of the midgut blood supply Infarction and gangrene of a part or all of the midgut may rapidly supervene

The diagnosis is made in neonatal life by the presence of vomiting and distention which depend for their severity on the location of the obstruction and grave disturbances of fluid and electrolyte balance Systemic signs of fever and leukocytosis usually indicate rapidly progressive gangrene of the intestine. During childhood and even early adult life partial and incomplete intestinal obstruction may be manifested by recurrent and intermittent abdominal pain with or without vomiting Occasionally minor degrees of incomplete malrotation such as a freely mobile cecum or an inverted cecum (arrest in the right upper quadrant) are found and are easily correctable

Successful operative therapy of obstruction and volvulus due to malrotations of the gut must be accompanied by accurate and detailed preoperative and postoperative management as in any case of intestinal obstruction

Meckel's Diverticulum and Persistent Vitelline Duct

Meckel's diverticulum is the remnant of the vitellointestinal duct. In early fetal life the intestinal tract communicates with the yolk sac. This patent communication is gradually obliterated. However the remnant of this yolk stalk, a thin cord or string of tissue may still in adult life connect the ileum to the umbilicus. Persistence of patency may occur at either end or throughout the entire length of the yolk stalk Patency at its attachment to the ileum leaves a short, blind pouch known to us as *Meckel's diverticulum* This condition is found in about 2 per cent of all persons Most of these diverticula are asymptomatic throughout life However, they may produce clinical disturbances in any of a number of ways. Obstruction of the small intestine may be produced when a portion of gut is occluded by a band representing the obliterated duct site of the diverticulum which stretches from the ileum (about 18 in from the ileocecal junction) to the umbilicus If the entire tract remains open it provides a communication between the lumen of the ileum and the umbilicus. This is a *persistent vitelline duct* its existence can be confirmed by x ray examination with radiopaque material Treatment is complete surgical excision and is uniformly successful. Patency of the distal end of the yolk stalk gives rise to a cyst or sinus at the umbilicus Secretions from the epithelial lining of the pouch distend the cavity to form a cyst if the umbilical opening becomes occluded. If it remains patent, a sinus is produced. Cysts may also occur in the midportions of the stalk if there is circumscribed patency

Inflammation of Meckel's diverticulum diverticulitis with or without perforation simulates appendicitis closely in both symptomatology and complications differing only in the location of the pain and tenderness. Perforation of the diverticulum and hemorrhage from it are complications of peptic disease of Meckel's diverticulum This is possible because *heterotopic foci of gastric mucosa* are not infrequently found in this vestigial structure Similarly bleeding peptic ulcer of Meckel's diverticulum may occur

Indeed, it is the most common cause of bright rectal bleeding in childhood Pancreatic tissue is also occasionally described histologically in Meckel s diverticulum Clinical symptomatology of diverticulitis consists of signs and symptoms of local peritonitis in the lower abdomen and of generalized signs of infection fever, and leukocytosis The preoperative diagnosis is usually appendicitis. Suspicion of Meckel's diverticulum is not infrequently aroused when there is otherwise unexplained rectal bleeding particularly in a child In such cases the diagnosis of a bleeding peptic ulcer of the mucosa in or near the diverticulum is usually substantiated. Less commonly the diverticulum forms the leading edge of an intussusception Fistula between the diverticulum and the umbilicus is even more rare

Therapy of the complications of Meckel s diverticulum is diverticulectomy It usually suffices to remove the diverticulum itself but occasionally a wedge of the ileum must be removed with it

Meconium Ileus (Mucoviscidosis)

Meconium ileus is a name which is given to just one facet of a perplexing generalized disease *mucoviscidosis* The disease consists of a disturbance of all cells which produce mucus, regardless of the organ system or epithelium involved The respiratory tract is prominently involved and renders the patient subject to *intercurrent pulmonary infections* which are frequently life taking. Although not primarily a mucus-secreting gland, the pancreas is particularly involved in both frequency and severity (thus the disease was formerly called *fibrocystic disease of the pancreas*) Its exocrine function is reduced, particularly the formation of proteolytic enzymes, and the glands are severely atrophic and inactive There is an increased amount of connective tissue in the gland, and it is palpably firm. Whatever pancreatic juice is delivered to the duodenum is abnormal. The mucus-secreting cells of the intestinal tract may be similarly involved Pathologic physiology which results from these anatomic and physiologic changes consists essentially of the accumulation in the distal portion of the small bowel of a very sticky, tenacious and rubbery material which obstructs the small intestinal lumen This is due to the absence of pancreatic digestion of intestinal contents. This abnormal meconium frequently cannot be removed from the gut at operation Rupture of the small intestine proximal to the meconium obstruction is not infrequent.

The diagnosis of meconium ileus is usually made early in life and although the differential diagnosis from other causes of obstruction is not always easy there are some characteristic findings Abdominal distention is usually maximal Radiologically an important diagnostic sign is the great variation in the size of the loops of dilated small bowel, and areas where the meconium is actually obstructing appear granular on the x ray film. Occasionally a hard mass of inspissated rubbery material can be felt through the abdominal wall.

Therapy of the meconium ileus obstruction facet of the disease *mucoviscidosis* has yielded very dismal results Multiple areas of obstruction offer no hope of survival For many years attempts were made to wash the material out at operation, both with saline and with proteolytic enzymes Little salvage was obtained by this technic Resection of the involved segments with primary anastomosis has led to uniformly sad results. Exteriorization of the obstructed segment in the Mikulicz *fashion* with subsequent resection appears to offer more hope The ileostomy is closed within a week. Adjunctive methods of treatment include the administration of pancreatic juice and other proteolytic enzymes orally and by instillation through the ileostomy These children require large doses of vitamins because in the presence of this marked pancreatic deficiency there is a malabsorption syndrome Antibiotics must be used generously to avoid fatal pulmonary infection. Nutritional care must be exacting, including oral and paren

teral proteins in abundance as well as a high caloric intake

BILIARY TRACT ATRESIA

Diagnosis

Atresia of the biliary ducts intrahepatic and extrahepatic is one of the relatively common causes of jaundice in the newborn infant. It must be differentiated from the other causes of jaundice in the neonatal period These are erythroblastosis fetalis, physiologic icterus of the newborn, the jaundice of severe infections and hepatitis The outstanding differential point in atresia of the biliary passages is the severe complete and unremitting degree of the icterus This is associated with totally acholic stools from birth and an accompanying large amount of bile pigment in the urine The associated prolonged prothrombin time is a result of both the concomitant cirrhosis of the liver and the failure to manufacture vitamin K in the intestinal tract without the presence of bile. The prolonged prothrombin time is responsible for an increased bleeding tendency

Treatment

Therapy of atresia of a portion or all of the biliary passages is necessarily surgical When explored, more of these children present types of atresia which are irremediable than which are amenable to anastomotic reconstruction The atretic disease may be entirely intrahepatic or may be of the extrahepatic passages exclusively In general, only about 25 per cent of all cases explored are amenable to available surgical procedures It is not advisable to operate on these infants until about the fourth week of life, at which time medical causes for the occurrence of jaundice can be proved or excluded

REDUPLICATIONS AND ENTEROGENOUS CYSTS

Duplications (reduplications) are structures of extremely varied size shape and length which consist of a cavity surrounded by a wall which closely resembles that of some portion of the alimentary tract. They may occur in the esophagus or stomach but the small intestine is by far the most frequent site Those which are elongated, as in the intestine are easily recognizable as duplications those which are small globular or round are usually referred to as *enteric* or *enterogenous cysts* Wherever they occur and whatever size they are they are *reduplications of the alimentary tract.* The epithelial lining of the cyst may not be similar to the structure to which it is adjacent. Those which contain gastric mucosa may be subject to peptic disease Almost all of these structures are closed at both ends

The symptomatology of reduplications of the intestinal tract varies greatly in nature and time of occurrence. They may not become manifest until adult life. Symptoms and signs depend upon the location of the cyst and its contents and may include anything from simple pressure on adjacent viscera to necrosis hemorrhage and peptic disease Treatment of these lesions is total excision This usually must be accompanied by excision of the associated piece of intestinal tract which is reduplicated Lesser procedures such as exteriorization of the lumen of the cyst are now outmoded In areas difficult for surgical intervention such as near the duodenum the cyst may be anastomosed to the normal bowel segment. Some duplications can remain in situ without compromising normal longevity

ANOMALIES RELATED TO THE UMBILICUS

Omphalocele

An omphalocele is a hernia of abdominal viscera into the base of the umbilical cord. Since the herniated contents are not covered by an abdominal wall, it may be considered an *eventration* The viscera are covered only by one layer of peritoneum and *one layer of* amniotic sac In all probability this defect

is due to an arrest of development of the abdominal wall at about the tenth week of fetal life when the extraabdominal viscera usually return to the coelomic cavity

The omphalocele may vary in size from a few centimeters to the entire width of the abdomen At least one-half of all cases of omphalocele are associated with other con genital malformations Occasionally a baby is born in whom the peritoneum and amniotic sac covering the omphalocele have already ruptured

Treatment of omphalocele is immediate surgical repair and should be carried out as soon as the diagnosis is made Delay merely invites superimposed complications, which may preclude success If the extruded vis cera can be returned to the coelomic cavity without undue pressure on the great vessels and on the diaphragm the operation may be done in one stage, closing the abdominal wall in layers However if the return of the extruded viscera will result in untoward symptoms such as dyspnea and obstruction to return flow from the lower extremities, then the operation must be completed in two stages The first of these is the under mining of skin flaps which are brought together over the extruded viscera. Subsequently in months to 2 years a second stage, in which the abdominal wall is recon structed in layers can be accomplished.

Hernia

An umbilical hernia is a fascial defect at the superior border of the umbilicus where this structure has been traversed by the blood vessels of the umbilical cord. It is a very common lesion seen more frequently in females than in males. Small umbilical hernias vanish as the growth of the rectus muscles proceeds usually becoming obliter ated by the age of two Larger ones should be repaired surgically as an elective procedure

Urachal Cysts and Fistulae

Urachal cysts and fistulae are remnants of the embryonic allontois If the entire tract persists, from the bladder to the umbilicus, a vesicoumbilical fistula exists If a portion of it is pinched off a urachal cyst remains In the former case there is a discharge of urine intermittently from the umbilicus, in the latter there is a tumefaction between the umbilicus and the pubis. Infection generally results It may be detected in adult life as a tumor or an abscess. Diagnosis of a urachal fistula is made by the extravasation of urine from the umbilicus and is confirmed by radiographic visualization of the tract with opaque materials Excision is almost always successful

Yolk-stalk cysts and sinuses have been discussed above with Meckel's diverticulum

DIAPHRAGMATIC ANOMALIES HERNIA AND EVENTRATION

Embryonic separation of the coelomic from the thoracic cavities occurs by the joining of two septa, one from the septum transversum ventrally and another from a septum derived from the dorsal mesentery This membrane is originally incomplete, allowing a communication through the pleu roperitoneal foramina (of Bochdalek) which apertures are later obliterated by a confluence of the pleura and the peritoneum The muscle of the diaphragm develops later between its pleural and peritoneal covers An embryologic arrest before these septa from the diaphragm results in a *congenital diaphragmatic hernia*, arrest after the pleura and peritoneum have fused, but before the muscle develops results in a *congenital diaphragmatic eventration* The latter is little more than a hernial sac surrounding abdominal viscera which have protruded into the thoracic cavity

By far the most common site of congeni tal diaphragmatic hernia is through the left pleuroperitoneal sinus of Bochdalek oc casionally a hernia exists through its counterpart in the right diaphragm permitting the liver to ascend into the chest. Congenital hernias of the esophageal hiatus and of the

retrosternal foramen of Morgagni are much less common

Congenital diaphragmatic hernias of the usual variety rarely possess a hernial sac The chest may harbor more abdominal viscera than does the shrunken *abdomen* These may include *stomach colon, sp* and *small bowel The left lung* may be (pletely compressed or even hypopl: True *esophageal hiatus* hernias co hernial sacs, which limit their ascent. short esophagus type of esophageal h

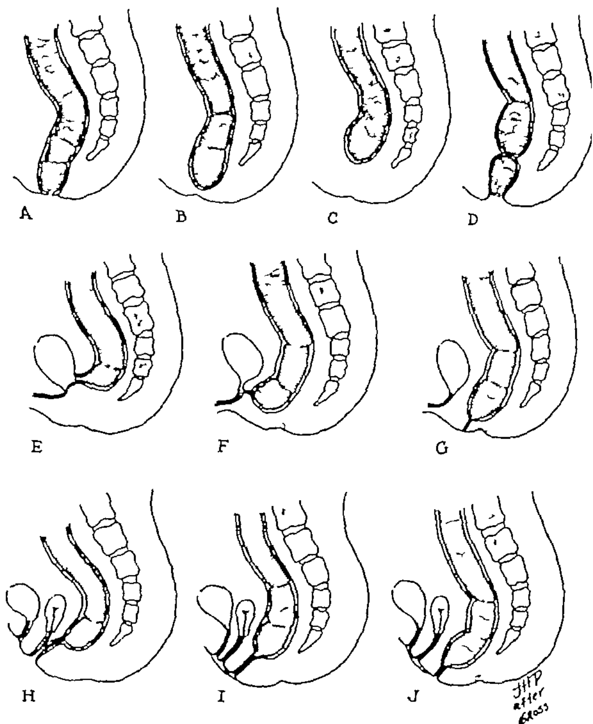


FIG. 41 Anorectal malformations. *A* Stenosis at the anus. *B* Imperforate anus with persist obstructing membrane. *C* Imperforate anus with rectal pouch ending blindly several centimet above the anus *D* Normal anus rectal pouch separated from anal canal by a membrane Male rectovesical fistula. *F* Male rectourethral fistula *G* Male rectoperineal fistula. *H* male rectovaginal fistula *I* Female rectofossa navicularis fistula. *J* Female rectoperineal tula

hernia is not a true hernia but rather represents a short gullet with the upper portion of the stomach beginning in the posterior mediastinum. Symptoms of congenital diaphragmatic hernias may be respiratory (cyanosis), owing to compression of the lung, or gastrointestinal which usually are vomiting and consequent failure to gain weight. Diagnosis can almost always be pinpointed by a roentgenologic examination. Treatment of congenital diaphragmatic hernias which are large enough to be productive of serious symptoms consists of operative therapy almost as soon as the diagnosis is made permitting, of course enough hours or days for proper preoperative hydration of the baby and deflation of its alimentary canal. Because adhesions between the coelomic viscera and the thoracic structures are almost always absent at this stage a transabdominal approach is desirable. Additionally any associated malformations of the intestinal tract can be simultaneously corrected. This differs with the approach in adults, where a transthoracic avenue is much to be desired. Expansion of the ipsilateral lung must be assured. Esophageal hiatus hernias should be treated preferably by a transpleural approach avoiding strangulation of the stomach and assuring reconstruction of the muscular hiatus. In eventration, the thin sac is excised, and the resultant edges of the muscular diaphragm are brought together in the reconstruction.

COLON, RECTUM, AND ANUS

Atresia and Stenosis

Atresias and stenoses occur in the colon proper and differ in no significant way from those in the small intestine except that they are much less frequent. The interesting malformations that occur at the anorectal end, however are quite common and may be of a serious nature.

In the five week embryo the genital and intestinal tracts terminate in a common cavity the cloaca. By the seventh fetal week these two tracts are separated by mesodermal structures. Soon afterward the urogenital tract is opened to the outside somewhat later the anal dimple ruptures onto the surface. Anomalies are caused by the arrest of this development which may occur at any stage. The nature of the defect is determined thereby.

Imperforate Anus and Fistulae with Genitourinary Tract

Imperforate anus of the simplest type, in which there is a very thin membrane, consisting of skin alone between the rectal cavity and the outside, is really a congenital anal atresia and the occluding skin is a persistence of the anal membrane. The term *imperforate anus* is applied broadly to developmental arrests of all degrees, including the most severe. Such defects include (1) the persistence of the anal membrane mentioned above (2) cases in which the rectal pouch is separated from the anal pouch by a complete septum (3) cases in which the rectal bulb ends anywhere from 1 to 6 cm above the anus, and (4) complicated types of imperforate anus associated with fistulae between the anorectum and the urogenital tract in addition to the atresia. In the male the fistulous tract usually is between the rectum and the membranous urethra or the bladder. In females there is usually a rectovaginal fistula (Fig 41).

The diagnosis of imperforate anus is usually made by simple postnatal inspection. If there is an anal opening in a proximal atresia, the symptoms suggestive of intestinal obstruction lead to further study. In imperforate anus associated with fistulae the passage of stool and/or flatus per vagina or urethra are diagnostic. Appropriate x ray studies with radiopaque materials confirm and detail the diagnosis. The position of the lowermost end of the rectal pouch is determined roentgenographically by measuring the distance between the termination of the bowel shadow and the skin. The infant is held in the upside-down position causing the gas to rise to the most distal portion of

the blind gut The possibility of associated congenital malformations elsewhere should be kept in mind

Atresias require immediate surgical relief. Stenoses, with or without fistulae are less urgent The choice of operation depends on the type of lesion and the condition of the patient In general, one stage reconstruction if feasible is preferred. Often however primary abdominal colostomy is a necessary first stage with the definitive repair postponed to a later date Simple strictures can be relieved with gentle dilatation Membranous imperforate anus can be treated by simple incision through the anal dimple More complex varieties of imperforate anus with or without an associated fistula, can be successfully operated upon through the perineal approach exclusively with gentle but adequate mobilization of the rectum and suture of the rectum to the anocutaneous line after pulling it through the lumen of the sphincter A combined abdominal and perineal approach is used for those malformations which end higher than 4 cm from the anal dimple Associated fistulae may be corrected at the same time The abdominal portion of the operation must be performed with great delicacy if neurologic complications of the lower urologic tract are to be kept at a minimum.

Hirschsprung's Disease (Congenital Megacolon)

Megacolon is a disease of the early years of life associated with a huge abdomen and infrequent bowel movements The colon is seen on x ray examination to be markedly dilated with a constriction ring at or near the anus Malnutrition, in the untreated child is a constant accompaniment of this defect The disease is characterized by the onset of obstipation in early infancy There is an associated maximal enlargement of the abdomen The child may have no bowel movements for days or weeks. The marked abdominal distention is accompanied by system[illegible]ymptoms of malnourishment and weak[illegible] distention is frequently severe enough to produce signs of pulmonary embarrassment While this congenital lesion has long been recognized the mechanism responsible for it has remained obscure until recently established by Swenson, in 1949 Prior to that time the defect was thought to be in the nervous control of the dilated bowel Swenson observed that the distended portion functioned normally after colostomy was done indicating that lack of motor function in the contracted distal bowel segment was responsible On histologic examination of biopsy material from this segment he found it to lack autonomic ganglia of the myenteric plexuses. This led to changes in operative therapy which have revolutionized the treatment of this disease.

Diagnosis is confirmed by scout film radiographic examination of the abdomen demonstrating the hugely dilated loops of colon Repeated enemas with plain water are dangerous because of absorption of the water with subsequently disturbed fluid and electrolyte pattern (water intoxication) Barium enemas are also not innocuous, since absorption of water from them may leave concretions in the dilated colon which cannot be expelled Atony of the distended proximal bowel can be demonstrated by careful fluoroscopic examination

Historically treatment of this serious condition resolved itself into (1) the use of parasympathomimetic drugs which temporarily stimulated the colon to evacuation, and (2) lumbar sympathectomy for the purpose of removing the atonic influence of the lumbar sympathetics of the colon previously believed to be of etiologic significance These procedures temporarily produced constriction of the colon musculature and bowel movements however they are at best, of only temporary value and do not remove the pathology

It has been adequately demonstrated that the *aganglionosis* varies greatly in extent Although usually confined to an area of the rectosigmoid colon it may be found to include a large part of the descending colon and occasionally the entire large bowel is

found to be aganglionic Treatment now consists in progressive resection of the congenitally diseased segment with immediate histologic examination by the pathologist As soon as a normal piece of colon wall is reported i e. found to contain normal myenteric Auerbach s plexus and Meissner s plexus the resection is terminated The proximal colon which is now found to be normal is either brought down to a residual piece of rectum to which it is anastomosed intraabdominally or as is more frequently the case pulled through into the perineum to complete an abdominoperineal resection and anastomosis to the anus. Care is taken not to disturb the bladder and the pelvic nerves These procedures together with meticulous attention to preoperative and postoperative care of these sick children, are productive of gratifying results

SUMMARY OF PRINCIPLES

1 The alimentary tract is formed early in embryonic life from a primitive tube extending from the stomatodeum to the proctodeum

2 Atresias (no hole interruption of bowel continuity) and stenoses (narrowing of the lumen) are the result of incomplete vacuolization of the primitive intestinal tract during the twelfth week of fetal life. The etiologic mechanism responsible for these anomalies is not well understood. Virus disease in the mother during early gestation may be responsible

3 Failure in normal formation of the lips, buccal cavity, and upper respiratory passages may lead to anomalies of the mouth of varying severity The most common are harelip and cleft palate.

4 Branchial remnants in the neck ears, floor of the mouth and pharynx are anomalies in development of the branchial grooves and arches. They are discussed in Chapter 14

5 Atresias and stenoses occur in the development of the esophagus. The most serious type of congenital anomaly involving the esophagus is tracheoesophageal fistula of which there are numerous varieties These must be detected early in life in order to avoid life taking complications particularly aspiration pneumonitis. After a few hours are taken for hydration and prophylaxis against pneumonitis one-stage surgical correction is undertaken

6 The most common congenital anomaly in the stomach is hypertrophic pyloric stenosis. It is most frequently seen in first born males during the second to sixth weeks of life The vomitus contains no bile, visible peristalsis of the stomach is seen through the abdominal wall a tumor of the pylorus may be palpable and roentgenographic evidence of almost complete gastric retention and of a thin stream of barium in the middle of the tumor make the diagnosis Treatment is by pyloromyotomy of Fredet and Rammstedt Meticulous preoperative and postoperative care is mandatory

7 Atresias of the small intestine are frequently seen and are most common at the terminal ileum Stenoses are also common particularly in

the duodenum Atresia of the duodenum is best treated by duodenojejunostomy rather than gastroenterostomy

8 Other causes of obstruction of the duodenum in the neonatal period are compression by tight superior mesenteric vessels and an annular pancreas Duodenojejunostomy is the treatment of choice.

9 Malrotation of the alimentary tract may be of varying degrees of severity may be compatible with long life or may result in the clinical diagnosis of intestinal obstruction in the neonatal period Intestinal obstruction may be caused by the mechanism of volvulus of the small intestine on a very narrow root or as a result of paraduodenal herniation behind the mesentery of the large bowel

10 Meckel s diverticulum is the remnant of the vitellointestinal duct. The complications of this vestigial structure include intestinal obstruction hemorrhage due to peptic disease in the diverticulum caused by heterotopic gastric mucosa and intussusception due to the presence of a mass Bright rectal bleeding in a child is frequently due to peptic disease in Meckel's diverticulum

11 Mucoviscidosis is a generalized congenital disease in which there is a disturbance of all cells which produce mucus regardless of the organ system or epithelium involved The respiratory tract and the pancreas are prominently involved Meconium ileus in the neonatal period is a part of this picture It is due to the presence of sticky material obstructing the small intestine this material is probably the result of defective pancreatic proteolytic enzymes Resection of the involved segment by the Mikulicz technic is recommended. Nutritional care must be exacting and intercurrent pulmonary infections must be avoided

12 Atresia of the biliary ducts may be intrahepatic or extrahepatic Jaundice from this congenital anomaly must be differentiated from other causes of jaundice in the neonatal period Only about one fourth of the cases of biliary tract atresia are amenable to surgical therapy

13 Reduplications of the alimentary tract may occur anywhere from the esophagus to the anus but are most common in the small intestine Symptoms may not occur until adult life Treatment is total excision wherever feasible

14 Omphalocele is a hernia into the base of the umbilical cord It must be treated surgically and where the abdominal viscera have lost their right of eminent domain multiple operations may be required to restore a normal abdominal wall

15 An umbilical hernia is a fascial defect at the superior border of the umbilicus Many close spontaneously larger ones must be closed surgically

16 Urachal cysts and fistulae are remnants of the embryonic allantois connecting the umbilicus and the urinary bladder Complete excision from umbilicus to bladder is curative

17 Congenital diaphragmatic hernias are most common through the

left pleuroperitoneal sinus of Bochdalek Abdominal viscera in the left chest cavity may compress the left lung and produce respiratory distress Congenital diaphragmatic hernias through the sinus of Morgagni and through the hiatus of the esophagus are less common Congenital diaphragmatic hernias are usually corrected through a transabdominal approach with repair of the diaphragm and search for other associated anomalies.

18 Imperforate anus is of several degrees and types The simpler ones are treated by a perineal approach Those in which the rectum ends 4 cm or more proximal to the anus must be corrected by a combined abdominal and perineal operation Associated fistulae between the rectum and the genital tract are corrected at the same time

19 Hirschsprung s disease (congenital megacolon) is due to an aganglionic segment of the large bowel Treatment is by resection of the bowel with immediate histologic examination, and anastomosis of good proximal bowel to or near the anus

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The Stomach and Duodenum

I Surgical anatomy and physiology
II. Peptic disease
  A Incidence
  B Etiology (1) predisposing factors (2) precipitating factors
  C Pathology
  D Diagnosis
  E Treatment and prognosis
  F Surgical treatment of ulcer and its complications (1) indications (2) types of operation (3) postoperative complications (4) end results
  G Differences in gastric and duodenal ulcers
III Tumors of the stomach
  A Carcinoma (1) incidence (2) diagnosis and differential diagnosis (3) pathology (4) treatment and prognosis
  B Sarcoma
  C Benign
IV Tumors of the duodenum
V Diverticula
VI Hypertrophic pyloric stenosis
VII Foreign bodies
VIII Gastritis
IX Wounds
X Syphilis

SURGICAL ANATOMY AND PHYSIOLOGY

The stomach is an expanded portion of the alimentary tract that lies between the esophagus and the beginning of the small intestine Its size and shape vary greatly with volume of its contents changes in adjacent viscera its functional activity and disturbances in tone secondary to pathologic changes in other viscera. The superior or cardiac orifice of the stomach is its junction with the esophagus The distal orifice is the entrance into the duodenum which is guarded by the pyloric sphincter The fundus is the expanded upper portion of the stomach which rises high under the left of the diaphragm The gastric cardia is largest part of its body and lies just be the fundus the vestibule is the lower port of the cardia and is the most depend portion of the stomach in the erect positi The pylorus consists of the antrum a terminally the pyloric canal, which is final 1 in of stomach The organ lies sentially in the left hypochondrium parti covered by the left rib cage but it n descend and fill most of the upper abdom

and even the lower abdomen when the viscus is markedly distended particularly in the erect position The posterior wall of the stomach forms the anterior surface of the lesser omental bursa, and here the stomach is in close relationship with the pancreas posteriorly and the transverse colon inferiorly At its left edge the greater curvature of the stomach is in contact with the spleen From the greater curvature of the stomach the anterior leaf of the greater omentum is continuous over the transverse colon as the gastrocolic ligament Beyond the colon it becomes the anterior leaf of the omental apron The lesser curvature on the right side of the stomach is continuous without any line of differentiation, into the right border of the esophagus It possesses a vertical and a horizontal segment, the junction being the *Incisura angularis* To the lesser curvature is attached the gastrohepatic omentum, within which lies the gastric arteries

The stomach wall consists of a highly vascular thick mucosa which slides easily over its submucosa, a pliable layer with great tensile strength The musculature of the stomach is divided into three layers an outermost, longitudinal layer· a middle circular layer· and an inner one in which the fibers pursue an oblique course The pyloric sphincter is a thickening of the circular layer

The stomach receives a very rich arterial supply from the celiac axis This trunk is the origin of the left gastric, the splenic and the hepatic arteries These vessels form two great arches which surround the stomach that at the lesser curvature consists of the right gastric artery (a branch of the hepatic artery) and the left gastric (or coronary) artery arising directly from the celiac axis. The greater curvature arch is contained within the layers of the great omentum and consists of the right gastroepiploic artery (from the gastroduodenal branch of the hepatic) and the left gastroepiploic (from the splenic artery) Tortuosity of these vessels permits great dilatation of the stomach The greater curvature is partially supplied by the short gastric vessels which arise from the splenic artery The gastric veins correspond closely to the arteries and are drained by the splenic and superior mesenteric divisions of the portal vein the left gastric vein anastomoses extensively with the lower esophageal veins.

The lymphatic supply of the stomach is abundant Mucosal and muscular lymphatic vessels ramify extensively in the submucosa and then are drained by lymph nodes Those of the lesser curvature and right half of the stomach carry lymph to the nodes in the gastrohepatic omentum and to the *porta hepatis* and along the right border of the esophagus Drainage from the fundus is to the paracardiac lymph glands at the left junction of the stomach and the esophagus and into the splenic pedicle along the *vasa brevia* Drainage from the pylorus is essentially into the inferior gastric glands in the gastrocolic ligament and omentum and into the subpyloric group of glands

The duodenum is the first portion of the small intestine varying in length from 8 to 12 in (duodenum literally means twelve) and is possessed of many unusual characteristics It extends from the pyloric valve to the duodenojejunal juncture at the ligament of Treitz, essentially in the form of a C It is more dorsal than any other portion of the intestinal tract, being retroperitoneal in position except for its anterior peritoneal investment. It receives the excretory ducts of the liver and the pancreas, both derivatives of the embryonic foregut. The first portion of the duodenum which turns slightly upward and dorsally lies at the level of the first lumbar vertebra and just to the right of it. The junction of the second and third portions of the duodenum also overlies the right renal vessels The anterior wall of the first portion of the duodenum usually is in contact with the fundus of the gallbladder The mediodorsal surface of the duodenum is in intimate relationship with the head of the pancreas. It receives the common bile duct and the main duct from the pancreas these ducts enter the duodenum

through the papilla of Vater At this point the duodenum lies directly upon the inferior vena cava being separated from it only by loose areolar tissue This second portion of the duodenum which is the vertical limb of the C is crossed by the transverse colon Thus the first and second portions of the duodenum are *supramesocolic viscera* whereas the third and fourth portions of the duodenum are *inframesocolic viscera* In the open arch of the C lies the head of the pancreas The third part of the duodenum, the lower transverse division, runs horizontally across the ureter, vena cava, and aorta at about the level of the third lumbar vertebra. The superior mesenteric vessels run downward over the ventral surface of this transverse division on their way to and from the root of the mesentery The fourth portion of the duodenum is its terminal 1 in which ascends to the level of the second lumbar vertebra and joins the jejunum at the duodenojejunal flexure, which is suspended from the posterior abdominal wall by the ligament of Treitz. Thus the duodenum is a relatively fixed organ by virtue of its attachments to the stomach the peritoneum which covers it, the excretory ducts of the liver and pancreas, the passage of the transverse colon ventral to it and the ligament of Treitz which suspends it from the dorsal wall

The extensive meandering of this small but highly important piece of gut makes it possible for a perforation of the duodenum to give rise to many different clinical pictures Thus it may be perforated into the free peritoneal cavity the lesser omental bursa, the pancreas itself or the retroperitoneal space The main arterial blood supply to the duodenum is from the pancreaticoduodenal divisions of the hepatic artery The superior pancreaticoduodenal is derived from the gastroduodenal branch of the hepatic artery and the inferior pancreaticoduodenal is the first branch of the superior mesenteric artery Lymphatic drainage is into the porta hepatis and into the nodes around the head of the pancreas and along the celiac and the superior mesenteric axes.

The function of the stomach is digestive in nature i e the breakdown of foodstuffs. The breaking down of complex foods is both mechanical and chemical Mechanically the churning action of the stomach due to the powerful musculature reduces the size of ingested masses thus exposing more surface area to its chemicals Chemically the stomach only digests protein foodstuffs This is true despite the fact that foods that enter the stomach have already been exposed to the amylolytic activity of the salivary juices. Hydrochloric acid and pepsin, the two substances produced in the gastric mucosa of the fundus by cells specifically designed therefor are the agents most active in the breakdown of protein The secretion of these two substances into the gastric lumen has a varying base line and is subject to great increases and profound changes under the influence of several factors The *psychic phase* of gastric secretion is the increased output of these substances initiated by higher centers. Thinking or anticipation of food or the smelling of pleasant food odors reflexly initiate a chain of events resulting in increased secretion of gastric fluids The efferent limb of this reflex arc is in the vagus nerves Such increased secretion may also be precipitated by emotions other than anticipation of food for example the tensions that exist in the typical peptic ulcer patient The *gastric phase* the excitation of secretion of the gastric glands by the presence of food in the stomach itself is mediated through *gastrin* a hormone manufactured in the mucosa of the antrum when food is present in that portion of the stomach When the contents of the stomach become very highly acid, this humoral factor then inhibits the antral mucosa from manufacturing more gastrin thus exerting a regulatory and protective mechanism against the presence of too much acid A third component is the *intestinal phase* in which *enterogastrone* a hormone liberated in the duodenum in response to the presence of acid chyme exerts a further inhibitory control

When the stomach has partially digested the food and reduced it to particles of smaller size it ejects its contents *intermittently* through the pyloric canal into the duodenum. In this most interesting portion of small intestine the food is now subjected to the products of secretion from the liver and pancreas, which are alkaline in nature and which contain elements that further digest protein foodstuffs as well as carbohydrates and fats These alkaline enzymes neutralize the gastric chyme and so tend to protect the duodenal mucosa from its erosive activity The activity of these enzymes will be discussed in greater detail in Chapters 33 and 34 It is appropriate to note that food entering the duodenum is usually acid in character and that leaving it is alkaline.

Methods of study of the stomach and duodenum are essentially (1) roentgenographic, in which changes in structure from the normal may indicate disease of these organs themselves or of adjacent viscera (2) chemical, i e. the analysis of the contents removed by aspiration from these viscera and (3) visual by means of gastroscopy

PEPTIC DISEASE

Incidence

Peptic ulcer occurs in both stomach and duodenum The latter is at least ten times as frequent as the former Ulcers in both sites have many characteristics in common and yet maintain certain differences, which will be discussed later in this chapter The fundamental cause of peptic disease is not known, although numerous contributing factors have been well established. It is *most common between the ages of twenty and forty* although it can occur at any age having been described in the neonatal period and in the aged. It is four times as common in males as in females and is characterized by exacerbations and remissions the exacerbations occurring frequently with the increased tensions of life and, peculiarly in some individuals in fall and spring of the year

Etiology

Predisposing Factors Although the lesion is usually a small ulcer in the stomach or duodenum the causes are general and include personality, environment, food habits, and possibly genetic influences There is thought to be a constitutional type (Stiller) which is endowed with an ulcer diathesis and is predisposed to the disease Individuals of this type are described as being tall thin, tense and often introverted they are markedy vagotonic However ulcer disease is seen in all body builds and in persons whose general demeanor does not conform to this description

Precipitating Factors The precipitating factors in peptic disease are considered to be stress and strain to which individuals are subjected in their total contact with other persons and life situations in the world Despite the advances in psychiatric theory complete understanding of mechanisms involved are wanting It has been suggested that irritant foods are productive of peptic ulcerations of the upper gastrointestinal tract it is more likely that such foods are irritating to a pre-existing ulcer

Pathology

Of the many pathogenetic considerations involved, the *acid pepsin factor* of the gastric secretion has been most convincingly demonstrated and exhaustively studied While it satisfactorily explains most of the observed phenomena, it is unlikely that it is the only factor involved In patients with duodenal ulcer *hypersecretion* is present, in terms of both *volume* and *acidity* Dragstedt and his associates have shown that the vagal component of the gastric acidity is particularly involved This is manifested by an increased volume and total acid secretion between meals and particularly at night. Inasmuch as the psychic activity during sleep is largely unconscious and dream associated, this provides some measure of physiologic

support of the psychogenic basis of peptic ulcer Other factors which have been adduced are infection and local vascular changes, but evidence of these is lacking

The most common site of peptic ulceration is in the first portion of the duodenum, which is subjected to the jet action of acid contents as they are ejected from the stomach through the pyloric ring into the first 2 in of duodenum Although most ulcers appear in this area others appear further down in the duodenum and in the gastric mucosa The essential lesion may be a mucosal defect in the active stage or a slow healing indolent ulcer it may later be in a healing stage In some cases all stages are found in the same ulcer or in associated ulcers The defect may be limited to the mucosa and submucosa or may have penetrated the full thickness of the wall If the *penetration* is walled off by adjacent peritoneum or viscera, the reaction is localized When the adjacent peritoneum and other visceral structures are unsuccessful in walling off the spillage of gastroduodenal contents a frank *perforation* exists Microscopically complete destruction of the mucous membrane is visible punched-out and undermined areas are characteristic. The infiltration of the surrounding areas with neutrophils is indicative of infection in the wall. Evidence of repeated episodes of activity and healing is the presence of heavy scars, in the extreme leading to *cicatricial stenosis* of the lumen

The pathologic physiology which leads to symptoms in peptic disease is easily demonstrable and consists of two outstanding factors (1) *hypersecretion* of acid-gastric juice (2) *hypermotility* The hypersecretion results not only in the presence of a higher acid content in the gastric juice but also in the secretion of a larger volume, particularly at night. The demonstration that these two factors play an important contributing role in peptic disease has been demonstrated clinically and experimentally beyond any doubt Laboratory methods employed in such demonstrations include the stimulation and or inhibition of gastric se

cretion by chemical methods, with an
out the resection of various portions
stomach and its nervous innervation
macologic evidence, the use of parasy
icolytic and parasympathicomimetic
has yielded corroborative evidence.

Diagnosis

The diagnosis of peptic disease is
on the symptoms and signs occasio
the pathologic physiology of *hyperse*
and *hypermotility* of the stomach.
prominent of these symptoms is a c
teristic type of *pain* which is epigas
position sometimes to the right of t
gastrium and sometimes substernal.
further described as *burning* in natu
casionally of a *gnawing* character,
bling *hunger pains* This description
gestive of a chemical basis of the pain
suggestive is the regularity and time r
to food ingestion. The distress quite
larly occurs 2 to 4 hr after meals wh
stomach has emptied itself The patie
ther relates that the distress is relie
ameliorated by the ingestion of food
ticularly milk or some other substance
has amphoteric properties, returning
when the stomach is empty The de
ment of a constant, sharp searing t
pain at a later date, unrelieved by the
of food, portends *penetration* and,
mately *perforation* since it represe
localized peritonitis The patient will u
state that the symptoms which brough
to seek relief are aggravated during p
of stress and are minimal during perio
inner calm There are tendencies t
spontaneous remissions and toward ch
ity the pains may have a definitely se
nature, being most prominent in the
and the fall

The very best objective sign of
ulcer is unequivocal x ray evidence
will consist of a *crater* in the mucosa
active stage and of a deformity due to
in the healing stage Diagnosis is fu
abetted by the determination of the vo
of night secretion which is markedl

creased during the period when ulcer is active A test meal may be given followed by the examination of aspirated gastric contents which will almost always reveal a high content of acid both free and total

Treatment and Prognosis

It is generally agreed by internists and surgeons alike that the treatment of peptic disease is essentially nonoperative in character A careful management in a tractable patient will yield satisfactory results in 88 to 92 per cent of such cases according to the experience of many groups Since the disease is systemic and environmental these factors must be corrected as well as the chemical changes in the stomach and the local mucosal defects *Psychiatric therapy* is frequently useful Although the effective medical management of peptic disease may require many changes in a patient's life including food intake social conduct, and even method of earning a livelihood of those persons willing to make the adjustment 90 per cent can live to normal life expectancy without surgical therapy

Medical therapy consists in combatting the effects of hypersecretion and hypermotility and comprises the following regimen (1) *Anticholinergics* are useful in diminishing the secretion of acid by the gastric mucosa. These substances interfere with the delivery of impulses brought to the stomach in the efferent part of the reflex secretion of gastric juice (2) *Diet* is controlled in such a manner that direct stimulation of gastric acid pepsin secretion is minimized. This is accomplished by the elimination of alcohol-containing liquids and highly spiced foods and by minimal intake of caffeine in any form The use of fats, if not otherwise contraindicated, is recommended in peptic disease since its presence in the stomach inhibits the formation of gastrin (3) *Alkaline* or *amphoteric substances* are useful to neutralize the accumulation of acid in the stomach which cannot be avoided The original Sippy powder of calcium carbonate is little used today because of undesirable renal effects of a high calcium intake in its stead magnesium silicates and aluminum hydroxide are found most useful (4) *Antispasmodics* are useful in reducing hypermotility most of these substances are in the belladonna hyoscyamus group or are synthetic preparations resembling them. (5) *Sedatives* are usually combined with antispasmodic medication these help reduce reflex gastric secretion (6) An attempt is made to change the undesirable habits of the patient in his response to stress situations of life

Surgical Treatment of Ulcer and Its Complications

Indications. The surgical treatment of peptic ulcer is usually limited to those cases in which nonoperative management fails and to the care of certain complications All physicians agree as to the indications for surgical intervention in ulcer disease These are (1) *perforation* (2) severe or recurrent *hemorrhage* (3) *cicatricial stenosis* and (4) *intractability* to nonoperative management. With reference to the first three there can be little room for difference of opinion The term intractability however does have different connotations Too often it is taken to mean inability to control the symptoms of a specific exacerbation of the disease It should be broadened to include inability permanently and definitively to control the condition so that the hazards of severe and life-threatening complications may be avoided.

The aim of surgical management of peptic ulcer is the permanent reduction of the gastric acidity to a level which will permit relatively normal gastric physiology without the recurrence of peptic disease A number of complications occur with peptic disease of the stomach and duodenum which make surgical therapy appropriate in some instances and mandatory in others (1) *Obstruction* at the outlet of the stomach due to *cicatricial stenosis* of an ulcer which has undergone repeated activity and healing, requires operative therapy in order to provide

relief from progressive and repeated vomiting and from its resultant deterioration in nutrition (2) *Perforation* of a peptic ulcer demands operative therapy for closure of the hole at the earliest possible moment. Although an occasional report has appeared in the literature of treatment of perforated ulcers by the nonoperative method of continuous gastric suction, this method of therapy must be looked upon as an expedient and not as a choice It may be justifiable if the patient is brought to the surgeon s attention many hours or days following the perforation or when competent surgical attention is not available Perforation may be free in which the hole in the alimentary tract extends into the peritoneal cavity or it may occur in the form of a slow leak, in which case there is a running battle between the soiling of the peritoneal surfaces and the ability of those surfaces to contain the chemical and bacterial insult. Occasionally a perforation on the posterior surface of the duodenum is walled off by omentum and by the pancreas and its capsule this is known as *forme fruste* a frustrated perforation A similar situation may exist when an ulcer threatens to perforate even on the anterior surface of the duodenum or from anywhere in the stomach In such cases the ulcer is found to have penetrated through the mucosa, submucosa, and muscular layers but peritoneal defenses are mobilized with only the peritoneum standing between the luminal contents and the free peritoneal cavity This stage of the pathology is referred to as a *penetrating ulcer* Many perforations occur without an antecedent history compatible with the diagnosis of peptic disease In World War II among service personnel this was true in 42 per cent of all perforations. (3) *Hemorrhage* is the third and frequently the most difficult complication of peptic disease to manage Certainly this occurrence requires the exercise of more judgment than do the other complications If it is known that the ulcer is in the duodenum the management of bleeding ulcer depends on the severity of the hemorrhage the age of the patient, and the previous history of the ulcer *Small hemorrhages* do not necessarily require operation However if a patient has had repeated episodes of bleeding *despite adequate ulcer management* the desirability of definitive surgical therapy must come under consideration *Massive bleeding* is much more serious and not infrequently causes death of the patient. *Age* is of some importance In the younger subject blood loss is better tolerated, and the normal elasticity of the bleeding vessel favors retraction and spontaneous cessation of hemorrhage Patients over the age of fifty are much less likely to stop bleeding and early operation is frequently mandatory In such patients failure to control bleeding despite energetic replacement or recurrence of *hemorrhage while therapy is still being maintained* usually calls for emergency operative intervention

If it is known that the source of the massive bleeding is from an ulcer in the stomach there appears to be no disagreement that the management of the case calls for operation at the earliest moment. The main reason for this lies in the fact that most ulcers of the stomach are on the lesser curvature, which is supplied by huge blood vessels which more often than not will not stop bleeding. (4) The fourth indication for surgical therapy of peptic disease is usually given as an *intractable ulcer* Theoretically this refers to an ulcer which has been treated by an adequate medical regimen and yet does not respond sufficiently well to permit the patient to continue his daily pursuit without serious difficulty However it also refers to the patient who is intractable and who will not adhere to a properly prescribed regimen It must be pointed out however that the patient may be as intractable after the operation as he was before Included in the definition of intractability must be those patients who are unable or unwilling so to change the order of their lives and possibly their occupations as to permit permanent

and definitive healing of the ulcer disease

Types of Operation Gastric resection for peptic disease is usually by the Billroth operation either number I or II In the former after resection of the acid forming part of the stomach gastrointestinal continuity is restored by a gastroduodenostomy In the Billroth II type the duodenum is closed, and gastrointestinal continuity is restored by a gastrojejunostomy In the patient with a marked psychic factor gastric resection may be accompanied by a bilateral vagotomy Gastroenterostomy combined with vagotomy is reserved for the elderly patient who may not be able to withstand the rigors of a gastric resection

Postoperative Complications. Early post operative complications include (1) peritonitis due to leakage either from the reconstructive procedure or from the site of duodenal closure (2) lesser omental bursa abscess (3) perigastritis, and (4) pancreatitis due to injury of the pancreas during the operative procedure. The most feared late postoperative complications are recurrence of peptic disease with a *marginal ulcer* and *recurrent bleeding* Marginal ulcers usually occur at the site of anastomosis of the stomach with the efferent loop of jejunum Recurrent bleeding usually results from the old ulcer in instances when it could not be resected and was therefore excluded and occasionally from a new ulceration

End Results. The end results of surgical treatment of peptic disease by gastric resection with or without vagotomy are in general, very good. Recurrent stomal ulcers should occur in no more than 5 per cent of all cases When the resection was done for the complication of a bleeding peptic ulcer recurrence of the bleeding has a higher incidence approximating 10 to 15 per cent. The "dumping syndrome" is a perplexing postsurgical complication seen most frequently when the efferent stoma is large Such a situation permits of rapid emptying into the jejunum, followed by symptoms of weakness perspiration and syncope It has variously been attributed to rapid absorption of sugars from the jejunum with a rapid evanescent hyperinsulin response and to fluid and electrolyte disturbances due to rapid dumping of liquid gastric contents into the jejunum Although the final word on the cause of this vexing problem has not been written the establishment of a relatively small efferent stoma (simulating the narrow pyloric canal) seems to be associated with a minimum of this troublesome sequel

When a subtotal gastrectomy is performed (75 to 90 per cent of the stomach) the patient may become a "gastric cripple" He is unable to gain weight or even to maintain preoperative weight, and may have a hemoglobin level chronically below normal This is probably associated with the absence of the intrinsic factor manufactured in the gastric mucosa and with the large loss of nitrogen and of fat in the stool. Reconstruction of gastrointestinal continuity by a gastroduodenostomy thus allowing the food to traverse the duodenum in some way changes the intestinal content so that loss of nitrogen and of fat in the stool is minimized.

Differences in Gastric and Duodenal Ulcers

Ulcerations of the duodenum outnumber those of the stomach by 10 1 However a gastric ulcer is a more serious disease in two important ways. First, the incidence of hemorrhage in gastric ulcers is greater than in a similar number of duodenal ulcers. Moreover as pointed out in the section above exsanguination from a gastric ulcer is a more serious threat than from the duodenal variety Secondly gastric ulcers which appear benign not infrequently are carcinomas The roentgenographic diagnosis of an ulcer of the stomach and even gastroscopic examination of that ulcer (if it can be visualized) are no assurance that the lesion is not in reality from the beginning, an ulcerating carcinoma of the stomach. This has been found true in actual experience, in 10 to 14 per cent of such ulcers. Even the

greater omentum and transverse colon and the pancreas and esophagus (2) *lymphatic spread* along the course of the lymph channels and nodes which drain the area of growth the porta hepatis, the pedicle of the spleen and the pancreaticoduodenal nodes are commonly involved with extension along nodes surrounding the celiac axis and the superior mesenteric artery the finding of metastases in the liver is most likely on the basis of lymphatic spread occasionally metastasis is first seen to a supraclavicular node in the left side of the neck (Virchow s) (3) carcinomatosis peritonei i e drop metastases these are commonly found early in the pouch of Douglas around the rectum and upon the ovaries, which seem to have a chemotaxis for malignant cells the latter have frequently been called *Krukenberg tumors* (4) distant metastases to lung bone, and brain by the *hematogenous route*

Carcinoma of the stomach must be included in the differential diagnosis of all lesions of the stomach and in all debilitating states and, therefore is a consideration whenever symptoms referable to the upper intestinal tract are presented

Treatment and Prognosis Treatment of cancer of the stomach is unsatisfactory from any point of view Until the cause of cancer is found the only hope of improving the curability of this dread disease lies in early detection, by some method better than now available The presence of carcinomatosis peritonei or extensive spread to adjacent or remote viscera precludes operative therapy By these criteria, only 70 per cent of patients upon whom the diagnosis of cancer of the stomach is made are operable i e acceptable for exploration. Upon exploration, only 60 per cent of those operated on are found to be resectable (resection technically feasible) thus, only 42 per cent of all patients in whom the diagnosis is made will have a part or all of the stomach removed Not all these are removed with any hope of cure some are done obviously and purposefully for palliation only Of those upon whom resection is accomplished 30 per cent are alive at the end of 5 years Thirty per cent of the 40 per cent that are resectable means that only 12 per cent of those in whom the diagnosis was made are alive at the end of 5 years

When operation is decided upon the patient must be prepared as adequately as possible in as short a period of time as possible It is well known that malignant disease of the gastrointestinal tract, most particularly of the stomach results in hypoproteinemia, malnutrition and anemia An attempt to restore protein levels toward normal particularly with whole blood is important (See discussion on protein metabolism in Chapter 5) The operative procedure consists in resection of the largest part or all of the stomach together with all grossly involved lymph nodes and areas of potential spread (within the limits of technical feasibility) and reconstruction of gastrointestinal continuity

Sarcoma

Sarcomas constitute only 2 per cent of all gastric malignant disease Metastases are usually by the hematogenous route *Lymphosarcomas* constitute the most frequent variety of sarcoma. They are *radiosensitive* and the prognosis is relatively good Leiomyosarcomas and fibrosarcomas have a poorer prognosis.

Benign

Benign tumors of the stomach account for 4 per cent of all neoplasms of the stomach Adenomatous polyps are the most common variety other benign mesenchymal tumors account for the remainder and include leiomyomas, fibromas, and lipomas When discovered by roentgenographic means, the therapy is surgical excision because malignancy cannot be excluded without histologic examination

TUMORS OF THE DUODENUM

Tumors of the duodenum are not common An occasional carcinoma of the mu

WOUNDS

Wounds of the stomach may be incurred by either penetrating or blunt trauma In penetrating injuries there are usually associated injuries of contiguous abdominal viscera and frequently of supradiaphragmatic structures Blunt trauma to the abdominal wall may result in extensive rupture especially if that structure is dilated with fluid and or gas The pathway of a sharp instrument which has penetrated the abdominal or thoracic wall and the trajectory of a high velocity missile must be studied in arriving at an early decision to explore the abdominal cavity When there is a suspicion of perforation or penetration of a stomach (or any other hollow viscus) exploration must not be delayed Examination of the posterior wall of the stomach through the lesser omental bursa must not be overlooked

SYPHILIS

Syphilis of the stomach is a rare occurrence It is represented by a large hard plaque in the gastric wall usually containing an ulcerated area in the mucosa and resembling peptic disease It is a tertiary lesion Symptoms are protean as they are with most gastric lesions The diagnosis is usually made by biopsy in a patient with a known positive serology The pathology does not differ from that of lues elsewhere namely a panvasculitis and a paravasculitis with marked round cell infiltration Therapy of this disease is essentially the therapy of the specific infection although resection of the stomach or bypassing the lesion by gastroenterostomy may occasionally be necessary for local reasons In general the patient with a gumma of the stomach is less debilitated and the disease is of longer standing than would be expected in carcinoma.

SUMMARY OF PRINCIPLES

1 The complex surgical anatomy of the stomach and duodenum brings these two structures into intimate relationship with other important viscera intraperitoneal and retroperitoneal supramesocolic and inframesocolic Diseases of the stomach and duodenum may affect contiguous structures and disease of adjacent viscera may affect the function of the stomach and duodenum

2 The extremely rich blood supply to the stomach makes it well nigh impossible to devascularize this structure during most surgical procedures the very abundant and extensive lymphatic drainage of the stomach is responsible for very early spread of malignant disease

3 The main function of the stomach is to initiate the process of digestion both physical and chemical the chemical digestion refers mainly to protein foodstuffs

4 Peptic ulcer of stomach and duodenum is a disease the fundamental cause of which is unknown although numerous contributing factors are well established The importance of the acid pepsin factor has been established experimentally and clinically beyond doubt The psychiatric components of this disease although poorly understood are equally well established

5 Duodenal peptic ulcers are far more common than gastric ulcers Most gastric peptic ulcers are on the lesser curvature

6 The essential lesion of peptic disease is an ulceration of the mucosa

diagnosis of carcinoma has been made must be meticulous with respect to correction of hypoproteinemia malnutrition and anemia

17 The most common sarcoma of the stomach is lymphosarcoma it is relatively radiosensitive

18 Benign tumors of the stomach must be excised for histologic diagnosis.

19 The only common malignant tumor of the duodenum is carcinoma of the papilla of Vater

20 Diverticula of the stomach and duodenum are usually asymptomatic They may however become involved by peptic ulceration

21 Foreign bodies of the stomach are of several types metallic usually ingested by children prisoners and psychiatric patients and trichobezoars and phytobezoars, hair and vegetable casts of the stomach Small foreign bodies may be watched fluoroscopically as they traverse the intestinal tract. Large foreign bodies, including the bezoars must be removed surgically

22 Hypertrophic gastritis is a disease characterized by a marked hypertrophy and redundancy of the gastric rugae A high gastric acidity can usually be demonstrated and the symptoms are usually those of peptic disease

23 Atrophic gastritis is usually associated with chronic dyspepsia, pernicious anemia, and carcinoma of the stomach

24 Phlegmonous gastritis is a disease of unknown origin and dismal outlook.

25 Wounds of the stomach may be incurred by blunt or penetrating violence Any suspicion of perforation makes exploration mandatory

26 Syphilis of the stomach is a tertiary lesion In comparison with carcinoma, the patient is less debilitated and the disease more chronic

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The Small Intestine (the Jejunum and Ileum)

I **Surgical anatomy and physiology (see Chapter 28)**
II **Congenital anomalies (see Chapter 23)**
III **Infections**
- *A* Nonspecific
- *B* Granulomas (1) regional enteritis (*a*) etiology and pathology (*b*) diagnosis (*c*) treatment (2) tuberculosis (*a*) etiology and pathology (*b*) diagnosis (*c*) treatment
- *C* Parasites

IV **Tumors**
- *A* Benign
- *B* Malignant

V **Intestinal obstruction (see Chapter 28)**
VI **Allergic disease and malabsorption syndromes**
VII **Wounds**

INTRODUCTION

The embryology congenital anomalies, and surgical anatomy and physiology of the jejunum and ileum are discussed elsewhere where they are most germane to the under standing of the subject then discussed For example the embryology is treated in detail in Chapter 23 surgical anatomy and physiology are dealt with in Chapter 28

INFECTIONS

Nonspecific

Infections of the small intestine are common from the neonatal period to the end of life though rarely are they of primary concern to the surgeon Many of these are nonspecific, which really means that the causative agent has not yet been determined others are specific and the responsible microorganisms can be isolated and identified Their principal interest here is in the differential diagnosis between them and surgical diseases of the small intestine Many infections of the small gut are manifested clinically by symptoms and signs which are not dissimilar to those of the acute surgical abdomen In the case of typhoid fever and its related diseases spontaneous perforation of the ileum results in an acute peritonitis In the group of diseases whose pathogens are more difficult of identification, an inflamed intestinal tract is found at exploration, more likely than not filled with both fluid and air and accompanied by varying degrees of *mesenteric lymphadenitis* These infections usually respond well to the use of antibiotics which are effective against the common inhabitants of the intestinal tract *Escherichia coli* and the microaerophilic streptococci as well as some of the specific pathogens (typhoid paratyphoid, etc.)

Granulomas

Regional ileitis is a disease of unknown cause and recent recognition which is characterized by a rather typical clinical onset and course, *occurring most commonly in the early decades of adult life* and presenting quite a characteristic pathologic picture. This picture consists of single or multiple areas of granulomatous infiltration which are rather sharply defined from adjacent *segments of small bowel, most commonly* seen in the terminal ileum, although the more proximal portions of the small bowel *and the right colon may be similarly* involved. The affected segments of gut may vary from a few centimeters to several feet *in length. The mesentery to the involved* segments is similarly diseased. The granulomatous encroachment upon the lumen is productive of inflammatory manifestations, followed by intermittent and partial intestinal *obstruction* with a tendency to the formation of *fistulae* between adjacent loops *of small bowel proximal to the obstructions*. Fistulae may also occur between intestinal loops and the anterior abdominal wall or *viscera of other systems* such as the bladder.

Diagnosis of regional enteritis may be *very difficult in the acute stage*. An acute localized peritonitis often leads to the diagnosis of appendicitis. Upon exploration a *hot acutely inflamed, markedly thickened* and friable ileum (or other segment of gut) or cecum is found. The appendix may or *may not be similarly involved. In the chronic* stage the affected individual presents complaints of *cramps* and *diarrhea* alternating *with periods of normal function over an* extended duration. The bouts of cramps and diarrhea are usually associated with abdominal tenderness. The patient may have low grade fever. Bacteriologic study of the stool reveals no specific pathogens. X-ray



FIG 42. *A* Regional ileitis in an elderly patient. The loop of ileum is fixed and immobile; its lumen is narrowed by the granulomatous thickening of the bowel wall. *B* Recurrent regional enteritis in a young man, *5 years after resection of 5 ft of* ileum and right colon. Note the furring of the mucosa and the incursion of the granuloma upon the lumen.

examination of the small intestine is usually most characteristic The granulomatous areas of small intestine are represented by very narrow strands of barium passing through them *the string sign* The disturbed mucosa is reflected by a *furred pattern* and there is a tendency to puddling of barium (Fig 42) Fistulae between loops and between a loop and the abdominal wall are occasionally demonstrated roentgenographically There may be seen intervening areas of normal bowel (*skip areas*) between the stretches of affected intestine

There is no completely satisfactory method of treatment for regional ileitis. Neither medical nor surgical therapy is always curative but surgical therapy is obligatory for some of the complications of this obscure disease Intestinal and systemic biotherapy have been found wanting being productive of only temporary and partial amelioration The antiinflammatory steroids are frequently helpful in reducing the violence of an acute exacerbation or in mitigating the severity of a recurrence Although it has been suggested that there is a psychosomatic component in the origin of this disease psychotherapy has offered no startling results.

Surgical management of regional enteritis is mandatory when (1) the disease is totally unmanageable medically with continuing diarrhea and malnutrition, the latter due to hyperperistalsis and malabsorption (2) obstruction occurs, relatively complete or partial and intermittent and (3) symptomatic fistulae are present Even after surgical therapy has been agreed upon by both physician and surgeon there is no unanimity of opinion relative to the merits of resection of the diseased bowel as against bypass procedures. In the acute stage it is the consensus that markedly inflamed gut should not be unduly handled and certainly not resected Under these conditions the diseased bowel is bypassed by a short-circuiting procedure usually an ileotransverse colostomy (in continuity although some prefer to perform it in discontinuity) At a later date when the diseased segment has been "cooled off" it may be removed by a second procedure Some surgeons are content to allow the diseased segment to remain *in situ* after the short circuiting procedure Both schools of thought report equally favorable results. Neither is perfect, since recurrences are common They may follow the initial episode of the disease by weeks, months, or many years. Frequently more and more proximal segments of bowel subsequently become involved Cases are occasionally reported in which repeated resections were deemed necessary resulting in patients who had but a few inches of jejunum remaining to be anastomosed to the right colon. Under such a condition the effects of treatment equal or even exceed those of the disease in that malabsorption of vital foodstuffs leads to inanition marasmus, and death In many patients the disease becomes burned out" after years of activity with no further progression, leaving a state compatible with normal life

Intestinal tuberculosis has diminished in incidence with the reduced general frequency of the disease in this country The pathology as in tuberculosis anywhere is that of a chronic granuloma with foreign body giant cells, caseation and healing by fibrosis.

As the disease becomes less common the diagnosis becomes more difficult to make The "doughy" feel of the abdomen in a patient with multiple intestinal complaints is probably no more characteristic of this disease than of any other granuloma The presenting symptoms are usually diarrhea and cramps, associated with malnutrition and continuous over prolonged periods. The isolation and identification of the specific pathogen from the stool is diagnostic. Intestinal tuberculosis is frequently associated with anal fistulae and ulcers, which have a characteristic dirty gray discharge and from which the active microorganisms can be isolated

Therapy of the disease is considered to

be nonsurgical and rests with the effective drugs now available Therapy in children with this disease includes the public health problem of detecting the source of unpasteurized milk

Parasites

Parasitic disease involving the small intestine is more common in the tropic and subtropic climates. Treatment is essentially medical It is mentioned here only because of obstructions of the small bowel produced by massive helminthic infestation in the Middle and Far East. When parasites are discovered during surgery for other diseases, postoperative medical measures for their eradication must be instituted Some worms will crawl through the most meticulous intestinal anastomosis into the free peritoneal cavity and even through the abdominal wall wound

Amoebic disease of the small intestine is far more rare than that of the right colon It is mentioned here only to be included in the differential diagnosis of other granulomas.

TUMORS

Benign

Tumors, in general are not common in the small intestine Of the neoplasms, most are benign these include the usual mesenchymal varieties, lipoma, leiomyoma, and fibroma. Benign adenomatous polyps are not infrequently discovered as a cause of bleeding or as the leading element of an intussusception. Some benign tumors of the small intestine have systemic manifestations These include (1) Osler Weber Rendu disease the congenital familial hemangiomatosis, which may result in massive hemorrhage (2) Peutz Jegher syndrome, familial polyposis of the intestinal tract with deposition of melanin pigment on mucocutaneous surfaces (particularly the vermilion border of the lips) (3) carcinoid tumors, or chromaffin body tumors. Chromaffin tumors or carcinoids are neoplasms arising in chromaffin cells in many places in the body They are not uncommonly found in the small intestine About one third of all cases must be considered malignant in that metastases occur As judged by longevity even in patients with metastases, the malignancy must be considered to be of a low grade variety Because the chromaffin cells have a special affinity for silver stains they are also called *argentaffinomas* These lesions may rarely produce systemic effects (hypertension) through the production of 5 hydroxytryptamine

Malignant

Carcinoma of the small intestine is most unusual with the exception of carcinoma of the papilla of Vater in the duodenum This lesion is discussed in greater detail in the differential diagnosis of tumors of the head of the pancreas Most malignant diseases of the jejunum and ileum are lymphosarcomas, arising in collections of lymphoid tissue which are in greatest concentration in the distal small bowel Other sarcomas are rare Specific diagnosis is rarely made in advance of surgical exploration These lymphosarcomas are usually radiosensitive Implantation of metastatic malignant tumors from elsewhere in the abdominal cavity into the surface of the small intestine is a not infrequent cause of intestinal obstruction.

INTESTINAL OBSTRUCTION

See Chapter 28

ALLERGIC DISEASE AND MALABSORPTION SYNDROMES

Allergic disease and the malabsorption syndromes are diseases of function which may affect the mucosa of the small intestine as well as of other tissues. They are mentioned here despite the fact that their therapy is nonsurgical, because they must be included in the differential diagnosis and evaluation of all diseases of the small bowel.

WOUNDS

Penetrating and perforating injuries of the abdominal wall as well as blunt trauma to this area of the body frequently involve the small intestine, because of the wide distribution of its loops throughout the coelomic cavity Penetration and/or perforation demand immediate exploration lest peritoneal soilage go unnoticed. Blunt trauma to the abdominal wall may result in extensive rupture of the small intestine especially if that structure is dilated with fluid and/or gas A serious injury involving the blood supply to the small intestine occasionally goes undiagnosed Reference is made to fall from a height Among the characteristic injuries of this type of violence those of the skeletal system (calcaneous acetabulum and pelvis, vertebral bodies) are best recognized Frequently the symptoms and signs of an acute abdomen are found after this type of injury All too often they are considered to be ileus, reflexive to the skeletal injuries In truth both at exploration and, unfortunately at autopsy the sudden deceleration when the individual hits the ground has resulted in a shearing action transmitted to the root of the mesentery with laceration of the mesenteric vessels from the aorta. Unless early exploration is carried out and this pathology recognized, gangrene of the small intestine and peritonitis may rapidly ensue

SUMMARY OF PRINCIPLES

1 Infections of the small intestine are common throughout life and are mainly of interest to the surgeon in the differential diagnosis of an acute abdomen

2 Nonspecific infections of the small intestine usually respond well to those antibiotics which are effective against the common flora of the intestinal tract

3 Regional enteritis is a disease of the early decades of adult life which is characterized pathologically by multiple areas of granulomatous growth of the mucosal surface a clinical picture of cramps and diarrhea, and, *in the severe cases partial and intermittent intestinal obstruction* Fistulae between adjacent loops of bowel and between the small intestine and the abdominal wall or the bladder are late sequelae

4 Diagnosis of regional enteritis is aided by the roentgenographic string sign which is evidence of the granulomatous encroachment upon the intestinal lumen and by evidence of formation of fistulae

5 There is no completely satisfactory treatment for regional ileitis Intestinal and systemic biotherapy produce temporary and partial amelioration the antiinflammatory steroids are helpful in reducing the violence of acute exacerbations and in mitigating the severity of a recurrence Psychotherapy is only occasionally helpful Surgical management is indicated when (*a*) the disease is totally unmanageable medically with continuing diarrhea and malnutrition (*b*) intestinal obstruction occurs (*c*) symptomatic fistulae are present The acutely inflamed intestine must not be unduly handled and certainly *not* resected A short-circuiting procedure (ileotransverse colostomy) is indicated A second stage may be performed for resection of the involved gut or if there is evidence that the disease is "burned out" the bypassed segment may occasionally be allowed to remain

in the abdominal cavity Repeat operations may be necessary when the disease recurs proximal to skip areas.

6 Intestinal tuberculosis is a vanishing disease in this country and may be treated adequately with the available effective antituberculosis drugs

7 Tumors of the small intestine are not common Benign lesions include lipomas, leiomyomas, and fibromas Familial polyposis (Peutz Jegher syndrome) and congenital familial hemangiomatosis (Osler Weber Rendu disease) are rarer diseases

8 The common malignant tumor of the small intestine is the usually radiosensitive lymphosarcoma The only relatively common carcinoma of the small intestine is that of the papilla of Vater

9 Allergies and malabsorption syndromes are diseases of function of interest to the surgeon because they must be included in the differential diagnosis and evaluation of all diseases of the small bowel

10 Penetrating and perforating injuries of the small intestine, proved or even suspected demand immediate exploration Blunt trauma to the abdomen and sudden deceleration by falling from a height may injure the small bowel by rupturing its wall or by severing its blood supply from its mesenteric root

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Appendicitis

I Surgical anatomy and physiology of the vermiform appendix
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  B Relations to adjacent viscera
  C Position
II Incidence and age distribution
III Etiology and pathology
  A Obstruction
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SURGICAL ANATOMY AND PHYSIOLOGY OF THE VERMIFORM APPENDIX

Embryology

The human vermiform appendix is a part of the midgut which ontogenetically and phylogenetically was a part of the embryonic cecum The anterior longitudinal cecal band (taenia coli) is continued over it. Its blood supply is derived from the ileocolic branch of the superior mesenteric artery its autonomic nervous innervation is the same as the rest of the midgut. The histology of the appendiceal wall differs from that of the cecum only in the thinness of its muscular layers It contains numerous accumulations of lymphoid tissue

Relations to Adjacent Viscera

The relationships between the vermiform appendix and other abdominal viscera vary widely The base of the appendix has a constant relationship to the cecum but the cecum may vary from its right lower quadrant station In a situation where the cecum

finds its final resting place in the right upper quadrant the anatomic relations of the appendix to the other intraabdominal viscera will be based on this somewhat less common arrangement

Position

The tip of the appendix may lie in one of many possible locations (1) It may lie free to touch the anterior abdominal wall (2) it may lie among the coils of the small intestine pointing medially (3) the tip may be pointing cephalad and be very near the fundus of the gallbladder or (4) pointing toward the pelvis and attached to the female adnexa, (5) attached to the urinary bladder (6) retrocolic and retroperitoneal (7) retrocolic between two layers of the parietal peritoneum or (8) attached to the obturator or femoral fossae

The appendix is possessed of its own mesentery the mesoappendix containing the blood vessels and lymphatics which supply it and a variable amount of fat The vermiform appendix may additionally be bound to the lateral wall of the cecum or to the parietal peritoneum by a fold of this latter structure known as *Jackson s membrane*

INCIDENCE AND AGE DISTRIBUTION

Appendicitis is the most common cause of a localized peritonitis, and therefore of the acute surgical abdomen. Although this disease occurs at any age in life its greatest incidence is in the first three decades, exclusive of the first 5 years in childhood It seems to be the consensus that acute appendicitis occurs far more commonly in the so-called civilized areas of the world than in those countries where life is somewhat more primitive

ETIOLOGY AND PATHOLOGY

Obstruction

Most cases of appendicitis probably begin with obstruction of the appendiceal lumen by a fecalith or by kinks Distention of the blind organ by secretions distal to the obstruction may become great enough to obstruct the veins draining it Additionally the appendiceal wall surrounding the obstructing fecalith may be so compressed that its blood supply is compromised When this occurs necrosis of the appendiceal wall is imminent This might be conceived of as an internal decubitus or a closed loop obstruction

Infection

Superimposed upon the *obstructive phenomenon* is *infection* the result of organisms present in the intestinal tract Inflammatory swelling occurs as with infection anywhere in the body Occasionally appendicitis occurs as part of a generalized enteritis Certainly it cannot be denied that in the picture of mesenteric adenitis in childhood the appendix is also found to be markedly inflamed the basic pathology however is the generalized enteritis. Some surgeons have claimed that associated intraabdominal infections may give rise to appendicitis This is difficult to deny and even more difficult to prove

Natural Course

The *natural course* of inflammatory disease of the vermiform appendix takes any one of several forms. It must be assumed that most mild cases of appendicitis are terminated early by the extrusion into the cecum of the obstructing fecalith or through conquest of the infection by defense mechanisms. Even appendicitis which has reached the active inflammatory stage can also subside without perforation of this viscus If, however as a result of ischemic necrosis or of virulent suppurative activity necrosis or perforation occurs, infection of the peritoneal cavity supervenes Thrombosis of the appendiceal vessels may produce early *gangrene* of the appendix. The marked inflammatory process within the appendix usually excites the defensive mechanisms in the area to wall off the process. This results in a

localized suppurative peritonitis, or *appendiceal abscess* It is seen frequently upon exploration that not only has the omental *apron found its way down to the right lower* quadrant and wrapped itself around the appendix, but also adjacent loops of small intestine have plastered themselves onto the inflamed viscus in such a way as to prevent the flow of its extruded contents into the rest of the abdomen The presence of the omentum and loops of bowel in the area wrapping themselves around the inflamed appendix gives rise to a palpable mass in the right lower quadrant the so-called *appendiceal mass* This mass may contain the defending omentum and gut without actual pus formation. More often the mass is compounded of the defensive structures surrounding an accumulation of fetid pus which communicates with the lumen of the inflamed appendix Occasionally when the defensive mechanisms cannot be adequately mobilized, the hole in the appendix is found to be spewing its contents into the free peritoneal cavity resulting in a generalized peritonitis. This dire complication is discussed in detail in Chapter 29 In young children because the defensive capacity of the omentum is less adequate this is more likely to occur

DIAGNOSIS

Symptoms (History)

The diagnosis of appendicitis is often the easiest to be correctly made in the practice of medicine again it may be the most difficult. The typical history from which there are many deviations, includes a series of symptoms usually presenting in chronologic order The first sensation of being unwell is not pain but rather *distress* usually localized to the periumbilical area This distress or *tormina* may be a sensation of bloating gnawing cramping or even a diffuse type of pain It is usually followed within minutes to hours by *nausea* and frequently by *vomiting* In a matter of hours, distress gives way to actual pain which is at first generalized but tends to become localized in the right lower quadrant of the abdomen It is described as a sharp or a strong dull pain with superimposed waves of cramping Constipation frequently occurs, although less commonly the initial symptoms may be associated with an episode of diarrhea. *Anorexia* is the rule Once the pain has shifted to the right lower quadrant and become more severe the patient may be aware of a generalized body warmth and will automatically flex the right thigh upon the abdomen to diminish the pain.

Signs

The physical signs in acute appendicitis are usually both systemic and local The generalized manifestations are those of acute infection and include fever of a moderate degree tachycardia, and a leukocytosis with a shift to the left. The local signs depend largely upon (1) the position of the appendix within the peritoneal cavity and (2) the stage of the pathology in its natural course The most typical findings are elicited when the tip of the inflamed appendix touches the anterior abdominal wall Palpation of the abdomen reveals marked *tenderness* over this site with much less or no tenderness elsewhere Such an abdomen reveals *muscle guarding* or *rigidity* in the right lower quadrant, which is absent on the left side This is due to a local nerve reflex from the irritated peritoneum to the muscles which overlie the area causing them to be spastic If some inflammatory exudate has leaked around to the left side or to the right upper quadrant, these areas may also be tender but as a rule they are less so than where the pathologic state begins. Deep palpation of the right lower quadrant with sudden release of the hand causes the inflamed appendix to rub against the peritoneum and to give rise to sudden pain at that area this is known as *rebound tenderness* and is a further sign of peritonitis *Localized peritonitis* of the right lower quadrant can further be elicited by deep palpa

tion on the left side of the abdomen thus pushing the cecum against the right parietal peritoneum When positive this is spoken of as positive *Rovsing sign* The site at which these signs of localized peritonitis are usually maximal occurs equidistant from the umbilicus and the anterior superior iliac spine an area spoken of as *McBurney s area* or *point*

If the tip of the appendix lies near the gallbladder the site of maximal findings will, of course be beneath the liver Similarly if the tip or any other part of the appendix, does not touch the anterior abdominal wall but lies elsewhere the findings will differ from those listed above. Thus, when the tip of the appendix lies in the retroperitoneum on or near the ureter or touching the bladder in the pelvis, the initial signs are those of urinary disease, with frequency and dysuria. In this event, the psoas muscle may be found to be in spasm. This is reflected in the positive *psoas sign* which is pain elicited when this muscle is put to use as by straight leg raising from the recumbent position Tenderness under these conditions is found to be in the flank or in the right costovertebral angle. The urine will reflect the presence of an inflammatory process, in or somewhere near it, by the many white cells found per high-power field. When the appendix lies partly or wholly in the midabdomen one s thinking is distracted from appendicitis and may be directed toward obstructions of the small intestine. *Rectal examination* should never be omitted in the examination of a patient with suspected acute appendicitis This may elicit a positive finding of tenderness if the tip of the finger can touch or approach the inflamed peritoneum

The temperature in appendicitis usually varies from 99 to 100 6°F in situations where the appendix has contained the infection If a fever of 102°F or more is encountered one must consider seriously the possibility of *perforation of the appendix* with either a *localized abscess* or *diffuse peritonitis* or other febrile disease The leukocyte count in acute appendicitis is elevated as a rule usually to from 8 000 to 20 000 with a *shift to the left* of 80 per cent or more polymorphonuclear cells. However cases are not rare where the white count is as low as 5 000 with 70 per cent polymorphonuclear cells, and a gangrenous appendix is found on exploration Exploration of course is made mandatory by the positive findings of localized right lower quadrant peritonitis. In such patients the gangrenous process is so rapid that the body has not yet had time to react with the usual leukocyte response *Dehydration* from excessive vomiting is seen only in neglected cases of appendicitis usually where perforation has occurred 2 days or more before the patient is seen by the examining physician

Diagnosis of acute appendicitis may be extremely difficult in the very young and in the aged In these extremes the abdominal findings are unreliable difficult to elicit in the baby and not infrequently masked by an inability to respond with the usual defensive signs in the aged At all ages, although hyperperistalsis may be heard in the early stages of the disease *ileus* is more common as time progresses

A summary of the diagnosis of this very common surgical condition is, indeed a summary of principles. The diagnosis of acute appendicitis is really the diagnosis of a localized peritonitis in the right lower quadrant of the abdomen. This is occasionally difficult and frequently easy to make So protean may be the manifestations of this very common disease that its diagnosis must be entertained as part of the differential diagnosis of almost any acute surgical abdominal problem Since this viscus is found to be perforated so frequently when it becomes inflamed it is better to err occasionally on the side of safety when an exact diagnosis cannot be made and explore rather than to be faced with a generalized peritonitis several hours later The differential diagnosis (*vide infra*) may be so complicated that a record of being correct may not exceed 80 to 90 per cent of all cases

DIFFERENTIAL DIAGNOSIS

A list of the diseases to be seriously considered in the differential diagnosis of an acute appendicitis is, of necessity a list of almost every disease which can produce abdominal pain. Most seriously to be considered however are (1) *diseases of the right urinary system* these include renal colic acute pyelitis, hydronephrosis and cystitis (2) *diseases of the right adnexa* in females, these include acute salpingitis, mittelschmerz, ruptured ovarian cyst of any type corpus luteum hemorrhagicum, and torsion of an ovary (3) *granulomatous diseases* of the terminal ileum, cecum, and ascending colon these include regional enteritis, enterocolitis, tuberculous typhlitis, and amebic disease (4) *acute gastroenteritis* these include those of *Staphylococcus Salmonella Shigella* and many virus types (5) *diseases of the extrahepatic biliary system* including cystic duct and common duct colic (6) *perforated peptic ulcer* of the duodenum or stomach with leakage of intestinal contents down into the right paracolic gutter (7) *neurologic diseases* of the spinal cord or of the peripheral nerves innervating the anterior abdominal wall these include the demyelinating diseases and the virus diseases such as herpes (8) *diseases of the right diaphragm* especially diaphragmatic pleurodynia associated with a right lower lobe pneumonia. In addition, such conditions as the exanthemata herpes zoster, metabolic acidosis purpura, and many others must occasionally be considered.

TREATMENT AND PROGNOSIS

The treatment of choice in acute appendicitis is the early removal of the diseased viscus, *appendicectomy* Unless it can be demonstrated that the attack of appendicitis has subsided by remission of symptoms and signs it is far safer to explore the abdomen than to risk the possible complications (*vide infra*) Before the antibiotic era it was accepted practice to operate immediately on a patient with the diagnosis of acute appendicitis, providing no appendiceal mass was palpable If a mass was palpated in the lower quadrant it was deemed wiser to treat the patient conservatively to allow the peritoneal defenses to wall off the inflamed organ, and to operate at a later stage when all signs had abated. This *interval appendicectomy* usually followed the acute episode by 6 or more weeks Since so many effective antibiotics are available now there is a growing tendency to operate even in the presence of an appendiceal mass. This is true for two reasons (1) if the mass is really an abscess (a collection of pus), the patient is better served if the pus is drained away than allowed to be absorbed or possibly to rupture into the free peritoneal cavity or even to become metastatic (2) if the appendiceal abscess is made up largely of loops of bowel, which is frequently the case, intestinal obstruction may supervene before the interval appendicectomy is accomplished If the diagnosis of a *generalized peritonitis* is made indicating the inability of the intraperitoneal contents to localize the infection, exploration is mandatory to close the hole in the gut which continues to contaminate the peritoneal surfaces Even in the aged and debilitated patient this principle holds true because while the peritoneal surfaces of all the abdominal viscera can do a remarkable job in combatting the insult from one episode of contamination, continued contamination by an opening in the gut is a far greater insult than the trauma of the operative procedure

The mortality rate of acute appendicitis should not exceed 0 1 per cent where the peritonitis is localized In generalized peritonitis, seen in very young and in aged and debilitated patients, the mortality rate continues to be 2 to 5 per cent despite antibiotic therapy Systemic biotherapy need not be employed when the appendix has been found not to be perforated It should be used wherever there is evidence of perforation and or spreading peritonitis Choice of biotherapeutic agents depends upon culture of

material removed at operation and on the principles discussed in Chapter 2

COMPLICATIONS

The sequelae of acute appendicitis are still numerous and serious That is to say the disease is such a common one that the morbidity which follows in its wake cannot be considered lightly The most common early complication of acute appendicitis is *generalized peritonitis* The reason for abdominal exploration is the attempt to control the generalized peritonitis by closing the hole in the gut For a detailed discussion of peritonitis and its total treatment, see Chapter 29 *Subphrenic abscess* an accumulation of pus in one of the spaces between the diaphragm and the liver is still occasionally encountered even in the antibiotic era. It is due to the leakage of infected intraperitoneal material along the right paracolic gutter into the subphrenic space usually the right posterior compartment It is characterized by a prolonged febrile course with pain in the right upper quadrant, elevation of the right diaphragm, and not infrequently a right sided hydrothorax. Treatment is effective drainage of the affected compartment. *Ascending phlebitis* along the course of the ileocolic vein, the superior mesenteric vein and the portal vein constitutes a serious complication When the portal vein is involved the term *pylephlebitis* is used to designate this disease It is characterized by a prolonged septic course associated with a high leukocytosis and shift to the left and with vague abdominal complaints Pylephlebitis is not infrequently followed by *intrahepatic abscesses* which may be very debilitating and extremely difficult to overcome Pylephlebitis following acute appendicitis is probably one of the important causes of portal vein thrombosis and its sequelae (see Chapter 33) *Pelvic abscess* particularly in the pouch of Douglas around the rectum, is still frequently seen as a complication of acute appendicitis with perforation and pelvic peritonitis. *Intestinal obstruction* may follow the attempt by the body to wall off the inflamed viscus in the right lower quadrant by the kinking of a loop of ileum which becomes trapped in the inflammatory mass and cannot escape from the mass. Intestinal obstruction may also follow the operation of appendicectomy if extensive raw surfaces are left in the mobilization of the terminal ileum and cecum or as a result of faulty technic, in which the ileum is kinked by improper suture

SUMMARY OF PRINCIPLES

1 The human vermiform appendix is a blind pouch which has no function and which frequently becomes infected. It may lie in the right lower quadrant its tip may touch the anterior abdominal wall, the bladder or the ureter or it may lie within loops of small bowel In partial malrotation of the gut the appendix will be found wherever the cecum comes to rest.

2 Appendicitis is the most common cause of a localized peritonitis may occur at any age in life and is more common in the so-called civilized areas of the world

3 Appendicitis may be initiated by luminal obstruction by a fecalith, by kinks in the appendix due to peritoneal investments and by infection associated with other processes within the peritoneal cavity

4 Many cases of acute appendicitis subside spontaneously

5 Most cases of suppurative appendicitis excite defensive mechanisms within the peritoneal cavity and are walled off by the omentum and loops of bowel These may form an appendiceal mass When the defensive mecha

nisms referred to are incapable of walling off a hole in the appendix, diffuse peritonitis is the result.

6 The diagnosis of appendicitis may be extremely easy on the one hand and very difficult to impossible on the other

7 The typical symptoms of appendicitis begin with a periumbilical tormina followed by nausea and vomiting the pain later becomes localized to the right lower quadrant Systemic symptoms are a low-grade fever and anorexia A silent abdomen (ileus) is the rule.

8 The typical findings of appendicitis include tenderness over the tip of the inflamed appendix, muscle guarding or rigidity rebound tenderness, positive Rovsing sign and a positive psoas muscle sign if the appendix lies retroperitoneally When generalized peritonitis already exists generalized tenderness and rebound tenderness throughout the abdomen will be found in addition to the localized signs

9 Diagnosis of acute appendicitis may be extremely difficult in the very young and in the aged.

10 The following diseases must be considered in the differential diagnosis of acute appendicitis diseases of the right urinary system diseases of the right adnexa in females granulomatous diseases of the terminal small bowel and cecum acute gastroenteritis of various types diseases of the extrahepatic biliary system perforated peptic ulcer neurologic diseases of the spinal cord or peripheral nerves, diseases of the right diaphragm, acute exanthemata metabolic acidosis and occasionally purpura.

11 The treatment of choice in acute appendicitis is early appendicectomy This is true today even in the presence of an appendiceal mass because of the availability of antibiotics which aid in localizing the infection Interval appendicectomy is performed much less frequently now than in the pre antibiotic age

12 Generalized peritonitis must be assumed to be due to continued soiling of the peritoneal cavity and makes exploration mandatory

13 The complications of acute appendicitis are generalized peritonitis subphrenic abscess, pylephlebitis intrahepatic abscess pelvic abscess, and intestinal obstruction

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The Colon

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A Surgical anatomy
B Surgical physiology
II Congenital anomalies
III Obstruction
A Clinical causes and types
B Pathologic physiology
C Diagnosis
D Treatment
IV Tumors
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B Polyposis (1) diagnosis (2) treatment
C Carcinoma (1) incidence and distribution (2) diagnosis (a) right colon (b) left colon (3) treatment and prognosis
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VIII Wounds
A Etiology
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BASIC CONSIDERATIONS

Surgical Anatomy

The colon or large bowel is 4 to 6 ft in total length (about one fourth the length of the small intestine) and lies in the abdomen in an inverted U position The ascending colon and descending colon are usually fixed to the parietal peritoneum and therefore to their positions within the ab-

dominal cavity but the transverse colon and the sigmoid are on mesenteries of varying lengths which permits considerable change in position The large bowel is recognized by its greater caliber the thicker wall and the presence of the three longitudinal *muscle bands (taeniae coli) on its surface* which together with the circular musculature, give the colon its segmental appearance (haustral markings) The colon is widest at the cecum and narrowest at its termination into the rectum at the level of the third sacral vertebral body

The colon begins with an expanded thin walled segment, the cecum normally placed *in the right lower quadrant of the abdomen* The terminal ileum opens into the cecum the orifice being guarded by the ileocecal valve which, when competent, permits passage of contents from the small bowel into the large but not in the opposite direction Arising from the cecum is the vermiform appendix, which was described in the previous chapter The degree of fixation of the cecum to the posterolateral parietal peritoneum varies and determines the possibility of volvulus of the cecum The cecum provides an important watershed in abscesses of appendiceal origin, often localizing such suppurative processes to the gutter

The ascending colon is immobile bound to the posterior abdominal wall by its peritoneal investment. Approach to the retroperitoneal structures is afforded by dividing the lateral peritoneal attachments and reflecting the colon toward the midline By this maneuver access may be had to the descending duodenum and the right kidney and ureter At the hepatic flexure the ascending becomes the transverse colon As the name implies, it lies just below the liver and in proximity with the duodenum and gallbladder

The transverse colon is suspended by its mesentery the transverse mesocolon, which is attached transversely and divides the abdominal cavity into *supramesocolic* and *inframesocolic compartments* The colon is also attached to the greater curvature of the stomach by the gastrocolic ligament which is the remains of the embryonic ventral mesogastrium This peritoneal fold extends beyond the colon and hangs freely from the anterior wall of the transverse colon as the greater omentum. The gastrocolic ligament *plus the posterior wall of the stomach* and the gastrohepatic ligament form the anterior wall of the lesser omental bursa, a subdivision of the coelomic cavity The inferior wall of the bursa is made up of the transverse mesocolon and the posterior by the parietal peritoneum over the pancreas and the vertebral bodies. The two omental bursae communicate only through the fora*men of Winslow a slitlike aperture between* the lateral free margin of the gastrohepatic ligament and the posterior abdominal wall

The transverse mesocolon stretches transversely from right to left and passes over the pancreas and the termination of the duodenum The duodenojejunal ligament (Treitz) is a triangular suspensory peritoneal fold, which gives attachment to the first part of the jejunum It lies to the left of the first lumbar vertebral body and is attached to the inferior surface of the transverse colon The superior mesenteric vessels also pierce the transverse mesocolon to the right of the duodenojejunal junction

The splenic flexure marks the transition from transverse to descending colon rises high in the left hypochondrium and is more acutely angled than the hepatic flexure It is attached to the diaphragm *by the phrenocolic* ligament. This portion too may be mobilized to give access to the spleen pancreas, and left kidney Passing downward, it is continued as the descending colon which is fixed to the left posterolateral abdominal wall by a peritoneal investment Its terminal portion the sigmoid colon, is suspended by a mesentery of varying length which as the name implies, throws it into an S-shaped curve It may be so mobile as to lie largely in the right side of the abdomen instead of its usual position in the center and in the left iliac fossa The left ureter lies dorsal to the normally positioned sigmoid

as these structures cross the pelvic brim This relationship is of importance in mobilization of the sigmoid colon in the surgical treatment of diseases to be discussed later in this chapter

The blood supply to the colon comes from two separate and distinct sources The cecum ascending colon and first half of the transverse colon are parts of the midgut and together with the rest of the midgut receive their blood supply from the superior mesenteric artery (successively the jejunal and ileal branches the ileocecal the right colic, and the midcolic arteries) The remainder of the colon is hindgut and receives its blood supply from the inferior mesenteric artery by way of the left colic, the sigmoidal, and the superior hemorrhoidal branches Projecting from the serous surface of the left colon particularly of the sigmoid division, are small processes, or sacs of peritoneum that appear much like lipomas each is provided with a blood vessel These *appendices epiploicae* are of defensive value in walling off inflammatory processes and perforations of the large bowel

The venous drainage of the colon, in general follows the arterial pathways and is tributary to the portal system The lymphatic drainage of the large bowel similarly is along the course of the arteries which supply the colon as they course toward the aorta. The importance of these anatomic facts is discussed later in this chapter

Surgical Physiology

Reference to Chapter 28 will show that the large bowel is really not one organ but two and has two separate and distinct functions. When small intestinal contents reach the right colon they are fluid in consistency The right colon, from the cecum to the midtransverse level performs the function of absorbing water and electrolytes thus desiccating the stool (The water and salts recovered in this part of the large bowel are the last two useful materials conserved by reabsorption from the alimentary tract.) The left colon is largely an excretory organ in that it removes from the body residue materials which have no useful purpose Water and electrolytes are absorbed in only a minor degree sufficient to produce progressive solidification of the alimentary wastes into a normally formed stool The descending colon sigmoid and rectum are well adapted to the handling of this more solid and sometimes hard material This natural adaptation takes the form of the increasingly heavy concentration of mucus-producing glands, whose product lubricates the lumen and provides freer passage than would otherwise be possible Also facilitating its excretory function is the heavier musculature of the left colon both the circular and longitudinal coats

The colon like the small bowel, is under the reciprocal influence of the two branches of the autonomic nervous system The sympathetic nerve supply (lumbar) is antiperistaltant and is in opposition to the parasympathetic supply (sacral) which is the positive innervation of the large bowel and produces maximal contraction of this organ The propulsion of the intestinal waste in a caudad direction is effected by the synchronous action of the circular and the very strong longitudinal musculature of the left colon.

CONGENITAL ANOMALIES

These are discussed in detail in Chapter 23

OBSTRUCTION

Clinical Causes and Types

Obstruction of the colon is common and is most frequently due to cancer in its descending portion Other less common causes are benign tumors, volvulus, intussusception, extrinsic malignant disease, pelvic inflammatory disease severe constipation with impacted stool and granulomatous strictures Each will be discussed in its appropriate section in this and the next chapter

Pathologic Physiology

Obstruction to the distal progress of stool in the colon results in either of two pathologic processes If the ileocecal valve is competent i e, will not permit the backward flow of material from the cecum and ascending colon into the ileum a closed loop is created between this valve and the obstruction distal to it. This loop continues to become distended with gas and secretions and may attain huge proportions As discussed in detail in Chapter 28 *closed loop obstruction* must be considered tantamount to a strangulating obstruction because the progressive distention, with no possibility of decompression from above or below will result in early perforation somewhere in this segment, as the thinning of the wall compromises its blood supply The diagnosis of closed loop obstruction then, requires immediate operative decompression by cecostomy or colostomy of the proximal ascending colon if the obstruction is on the right side or by transverse colostomy if the obstruction is on the left side

If the ileocecal valve is incompetent and permits the reflux of right colon contents into the terminal ileum the patient s illness will be less acute and more prolonged The symptoms under these conditions will resemble those of obstruction of the lower small intestine Indeed, the pathologic physiology is then the same as if the obstruction were in the terminal ileum with distention of that part of the gut and accumulation of fluid and air proximal to it. Differential diagnosis between large bowel obstruction with an incompetent ileocecal valve as opposed to obstruction of the terminal ileum is frequently difficult.

Diagnosis

The diagnosis of large bowel obstruction must be made before the reason for that obstruction is determined This is to say by the time obstruction of the large bowel has occurred it is urgent to relieve the obstruction rather than to treat the possible cause such as cancer diverticulitis or granulomatous stricture The diagnosis in large bowel obstruction with an incompetent ileocecal sphincter is understandably delayed, in that distention occurs more gradually and therefore over a longer period of time as opposed to the rapid distention in high small intestinal obstruction. Closed loop obstruction of the large bowel with maximal distention of this viscus results in marked abdominal pain and tenderness in a patient who is obviously acutely ill The pain frequently is greatest in the right lower quadrant, where distention of the cecum (the widest and thinnest walled part of the bowel) is maximal. The diagnosis of obstruction of the large bowel is suggested by the history given by the patient, as detailed later in this chapter of progressive constipation, obstipation, alternating constipation and diarrhea, the passage of bright blood and failure to pass flatus. Antecedent history of known diverticulosis or the availability of previous x ray films showing marked redundancy of the sigmoid may frequently make the diagnosis easier

Treatment

The *treatment of obstruction* of the large bowel is basically the same as the treatment of intestinal obstruction under any conditions However it must be emphasized and is worthy of repetition that the emergency is the decompression of the large bowel by a procedure which attempts to do no more than just that Definitive *treatment of the cause of the obstruction* is postponed until the patient has recovered from the physiologic disturbances occasioned by it essentially by adequate decompression and replacement of fluids after which further diagnostic studies may be carried out.

TUMORS

Polyps

Polyps of the colon, as elsewhere, are mucosal excrescences of various shapes

composed of normal and abnormal mucosa and mucosal derivatives A simple polyp has normal mucosa covering its entire surface An adenomatous polyp is one at the tip of which the mucosa has the structure of a neoplasm These tumors are believed to be benign originally but have a decided tendency to become malignant A polyp with a long stalk of normal mucosa is said to be *pedunculated* one that lies flat on the mucosa without a stalk is said to be *sessile* Fortunately the pedunculated ones are more common than the sessile the former are more easily removed, and malignant disease in them is less frequent. Polyps may be single or multiple at times the entire surface of the bowel may be studded with them (*vide infra*) The most common site of polyps in the large bowel is the sigmoid colon

The diagnosis of polyp of the large bowel is usually made on the history of bright blood in the stool and x ray visualization of a space-occupying shadow Occasionally the diagnosis of polyp is made with no antecedent history on routine barium study of the colon. Less commonly a polyp may grow large enough to produce symptoms other than bleeding such as intermittent obstruction, intussusception, or volvulus.

It is generally agreed that all polyps of the colon should be removed because of the danger of malignant degeneration. All such specimens must be carefully examined by the pathologist, since without such examination it cannot be determined whether malignant changes have taken place Occasionally these growths can be removed through the sigmoidoscope If not within the reach of this endoscopic procedure, they must be approached transabdominally through a colotomy Since multiplicity of polyps is common, the colon should further be examined for additional lesions at operation with the use of a sterile proctoscope Histologic study may reveal malignant changes in the tip of the growth which do not extend to the pedicle Under these circumstances simple polypectomy may suffice. If however, the base of the polyp exhibits changes that are considered to be malignant in nature segmental resection of that portion of the colon is necessary If lymph node metastases are present a more radical operation must be performed, as will be discussed later in this section In general sessile polyps are a greater cause for concern than are pedunculated ones because they are more often found to be carcinomatous and also because extension to the surrounding mucosa occurs more readily

Polyposis

Polyposis of the colon is a term used to designate a specific disease which is more than the mere presence of a single or several polyps in that organ It is characterized by a profound tendency for the mucosa of the large bowel to produce innumerable polyps of every variety and size This disease is a distinct entity familial in nature and of hereditary origin The patient usually comes for examination because of repeated bleeding from the rectum Digital examination reveals a roughened mucosa with palpable polyps, even at the level of the rectum Proctoscopic and sigmoidoscopic examination confirms the presence of many hundreds of these lesions. Roentgenography reveals that the entire colon is similarly involved One form of familial polyposis of the colon is associated with the finding of melanin deposits at the mucocutaneous junctions, particularly of the lips and mouth. This combination is referred to as the *Peutz Jegher syndrome* and it may be associated with polyposis of the small bowel.

There is only one treatment for polyposis of the colon, and that is total colectomy If the mucosa of the rectum is involved down to the anus, there is no alternative but to perform a combined abdominoperineal resection with permanent ileostomy If an inch or two of rectum is relatively uninvolved, an ileoproctostomy may be done, with frequent postoperative proctoscopic re-examinations and fulguration of polyps that may be seen to develop subsequently This drastic

approach to this disease is mandatory because in every case in which it is not done carcinoma develops in at least some among the myriad of lesions

Carcinoma

Adenocarcinoma of the large bowel is a common gastrointestinal cancer and parallels the age incidence of cancer in general although it is not uncommon in younger persons including those in the second and third decades of life As might be suspected from the fact that the mucus-producing glands are found in greatest numbers in the terminal left colon, this segment of the large bowel presents the largest incidence of adenocarcinoma. The rectum rectosigmoid and sigmoid colon account for 65 per cent of all cancers of the large bowel The cecum accounts for 13 per cent the ascending colon transverse colon descending colon and the two flexures each account for 4 to 5 per cent of all malignant lesions of this organ

Reference has been made to the differences between the right and left halves of the colon as to their embryologic origins, blood supply and function Corresponding differences are observed in the pathology diagnosis and management of carcinomas arising in the two segments.

Cancers of the right colon tend to be medullary fungating ulcerated and vascular The presenting symptom of such lesions, particularly of the cecum is either *progressive anemia* of unexplained origin vague digestive disturbances of the extragastric variety or the palpation of a mass in the right lower quadrant Because of the large diameter the distensible walls, and the liquid consistency of the contents at this level obstructive signs are uncommon

In the left colon particularly the sigmoid and rectosigmoid carcinoma tends to be scirrhous in type and to encircle the bowel It is first manifested by a characteristic train of symptoms and findings The patient presents himself with a history of having passed *bright blood* from the rectum or with a history of a *change in bowel habits* This change in bowel habits is usually in the direction of a progressively increasing constipation with the passage of pencil sized stools There may be *alternating periods* of *constipation* and *diarrhea* This may be due to the use of cathartics to obtain relief or to the ulceration of the lesion permitting *intermittent decompression* In the extreme *obstipation* is complained of namely no return of stool with the taking of an enema and of course no relief from the sensation of distention Not infrequently the patient s only presenting complaint is pain in the right lower quadrant of the abdomen This is due to the distention of the cecum and peritoneal irritation overlying it, when the ileocecal valve is competent It is under such conditions that a closed loop obstruction develops, as discussed earlier in this chapter Malaise and cachexia are very late symptoms

The sequence of examination is logical and well standardized when there is any suspicion of bowel cancer *Digital examination of the anus and rectum precedes all else* A large number of cancers of the terminal bowel can be felt by the examining finger *Anoscopic proctoscopic* and *sigmoidoscopic examination* of the mucosa of the distal gut follows immediately thereafter after proper preparation If a lesion is seen *biopsy* is performed at that time Histologic confirmation is definitive If no lesion is seen in the terminal 25 cm of colon examined *roentgenograms* are made of the large bowel The roentgenologist may be able to demonstrate a lesion beyond the reach of the sigmoidoscope and multiple areas of involvement may be revealed He must be careful not to force contrast medium beyond an obstruction for fear of creating a closed loop obstruction A picture of the bowel with a double contrast (air) is desirable *Exfoliative cytology* the examination of secretions and washings from the colon may reveal malignant cells even when a mass is not visible Whenever there is no obvious reason at the anorectal junction for blood in the stool exfoliative *cytologic examination*

is advisable The diagnosis of cancer of the bowel is occasionally not made until the malignant tumor results in *perforation* of this viscus into the free peritoneal cavity or into a viscus such as the bladder or into the retroperitoneal space Palpation of the abdomen will occasionally disclose the *mass* in the bowel and occasionally even its hepatic metastases The latter may be the earliest clinical manifestation. Rarely metastatic involvement of the vertebral bodies (via Batson's veins) is detected

The treatment of cancer of the colon is surgical extirpation Preoperative preparation includes intestinal antisepsis cleansing of the bowel from below restoration to an optimal nutritional state and the correction of hypovolemia and anemia by administration of whole blood Cancer surgery means wide excision of the lesion together with the blood supply to that segment of colon and all the lymph node–bearing area that is technically feasible In general, this means a right hemicolectomy for lesions of the right colon and a left hemicolectomy for lesions of the descending colon, sigmoid and recto-sigmoid portions Malignant tumor of the transverse colon requires resection of the entire bowel supplied by the midcolic artery and its lymph node–bearing area. The presence of lymph nodes in the mesentery or along the aorta and even some metastatic involvement of the liver is not a contraindication to such wide resections because prognosis for carcinoma of the colon, in general is much better than lesions of other organs, such as the stomach. Occasionally hepatic metastases have been observed to disappear in ensuing months and years, after resection of the primary lesion. The biology of cancer is unpredictable There are some tumors of the colon which metastasize very early when the primary lesion is very small. Others grow to a very large size and yet metastasize very late Operability and resectability of cancer of the colon, in general is much greater than that of the stomach Five year nonrecurrence rates vary from 40 to 80 per cent of all cases seen. Longevity is limited by (1) local invasion of adjacent viscera, (2) distant metastases and (3) extensive hepatic involvement. The occurrence of multiple malignancies of the colon at one time and at wide intervals of time are not uncommon When this type of pathology is encountered the relatively good outlook is somewhat dimmed

Mesenchymal Tumors

Malignant lymphosarcomas of the large bowel are occasionally seen but they are rare Benign mesenchymal tumors including fibromas, lipomas and leiomyomas, are occasionally encountered in the wall of the large bowel

DIVERTICULITIS

Incidence and Etiology

Diverticula are outpouchings of the mucosa and submucosa through the muscular wall of the gut usually on the mesenteric side Sites of predilection are the apertures in the muscular coat of the wall where perforating vessels enter it. Although they occur at any age in life diverticula are found most frequently in middle age and in both sexes. Roentgenographic surveys reveal that 10 per cent of all human beings over the age of forty have some diverticula. The most common site for diverticulosis is in the sigmoid colon.

Pathologic Physiology and Anatomy

Since diverticula communicate with the intestinal lumen they are frequently filled with fecal contents *Inflammation* and *edema* of the mucosa at the neck of the diverticulum may result in *obstruction* The diverticulum may then become infected, with the usual resultant inflammatory process in and around it The inflammatory process may produce (1) spasm of the involved colon (2) spasm of adjacent bowel (3) localized peritonitis, (4) perforation with local or generalized peritonitis, and (5) systemic signs of an infection Localized peritonitis due to perforation of a diverticulum may

result in a mass which may so constrict the bowel that the differential diagnosis between this disease and neoplasm is very difficult not only on roentgenographic examination, but at the operating table with the lesion under direct vision.

Diagnosis and Differential Diagnosis

Uncomplicated diverticulosis is asymptomatic. The diagnosis of diverticulitis is based on the history of *cramps* in the lower abdomen, occasionally with changing bowel habits, usually *constipation alternating with diarrhea* there is frequently passage of *bright blood* and there may be systemic symptoms of fever with its associated signs of a *generalized infection* leukocytosis and toxemia. The differential diagnosis between diverticulitis and neoplasm of the bowel is frequently difficult and a source of great concern The diagnosis of spasm due to diverticulitis is usually made when the bowel is observed to relax during fluoroscopic examination In carcinoma of the colon, no such relaxation is seen at any time once the obstructing shelf has been observed. Unfortunately adenocarcinoma of the colon *is not infrequently found in an area of diverticulitis* The symptoms of the two diseases may overlap so much as to confuse the differential diagnosis. When this is true *exploratory biopsy* and *examination* under direct vision are indicated, because barium enema and sigmoidoscopic examination will fail to differentiate between these two diseases

Treatment, Prognosis, and Complications

Diverticulosis without the presence of inflammatory disease requires little if any treatment. Diverticulitis frequently responds well to medical management of (1) low residue diet, (2) antispasmodics, and (3) biotherapy The indications for surgical therapy in diverticulitis are (1) recurrent attacks of inflammation (2) repeated hemorrhage (3) unremitting spasm, (4) the inability to differentiate the disease from cancer (5) obstruction of the colon and (6) the complications of the disease

Complications of diverticulitis may take any of several forms *Peridiverticulitis* and narrowing of the lumen of the bowel may produce obstructive signs For this reason alone operative therapy is indicated. *Perforation* in diverticulitis may be acute subacute or chronic Thus, a chronic abscess may exist, and the signs of localized peritonitis may grow over a period of days and weeks in free perforation an acute surgical abdomen may suddenly occur The formation of *fistulae* between the diverticulitis and adjacent viscera is much more common in this disease than in cancer of the colon. These abnormal communications may exist between the colon and the ureter or bladder or between the colon and a loop of small bowel, or occasionally between the colon and the female genitalia.

The sequence of measures in the treatment of the complications of diverticulitis is dictated by the nature of the complication and by the fact that postoperative disturbances following surgical attack for diverticulitis are far more numerous and severe than *after the same operations for cancer* These untoward postoperative sequelae are usually in the nature of *extensions of infection* (most frequently *peritonitis*) and *disruption of anastomotic lines* Because of this, the first stage in resectional therapy for diverticulitis is usually *proximal decompression by transverse colostomy* A varying length of time is allowed to elapse until the local and generalized symptoms of the inflammatory disease have abated This may vary anywhere from 1 to 6 months Resection and anastomosis are then accomplished at a second operation and closure of the colostomy at a third stage Identification of the left ureter in resections for diverticulitis is important and is best done by the preoperative placing of a ureteral catheter in retrograde fashion Very careful histologic examination of the specimen is required to rule out any associated malignant disease

Chronic nonspecific ulcerative colitis is a disease of youth and early adult life particularly in females constitutionally asthenic characterized by a profound inflammatory reaction in the colon and by severe local and generalized symptoms and signs Prominent in the manifestations are exacerbations and remissions of *diarrhea* and the passage of *pus blood and mucus per rectum* associated systemically with anemia, toxicity and cachexia. Complications in the diseased segment of the colon and in other remote structures are common.

Etiology

The cause of nonspecific or idiopathic ulcerative colitis is unknown Prominent among the theories of its causation are (1) a specific microbe which has never been isolated (2) disturbance in enzymes of the pancreas or in sensitivity of the bowel mucosa to them (3) disturbed nervous innervation of the colon (4) emotional factors. Although it is possible that secondary infection of the colonic mucosa is a part of the disease or even that the diseased mucosa is aggravated by intestinal enzymes and it is certain that psychosomatic illness accompanies this disease none of these pathogenic possibilities has been established as the primary factor

Pathology

The disease may involve all coats of the bowel wall The mucosa is primarily involved It is grossly *inflamed* and extensively *ulcerated* The ulcers are ragged, undermined, dirty-looking, and indolent. They may be *confluent* and involve most of the mucous membrane, which is replaced by a friable granulation tissue which bleeds freely Proliferation of this inflammatory tissue results in *polypoid elevations* which like the spontaneous polyps of the colonic mucosa, are believed to have a marked propensity toward malignant degeneration In contrast with the granulomatous disease *regional enteritis* which may also involve the colon there are no fistulae to other loops of bowel or to adjacent viscera or the cutaneous surfaces The muscle layers lose their healthy appearance and are in some places replaced by fibrous tissue The segmented character of the bowel is lost in the subacute and chronic stages and haustral markings are absent. The loss of haustral markings is responsible for the "lead pipe" appearance of the colon in the roentgenogram

Diagnosis

The course and severity of ulcerative colitis varies greatly The usual story is one of chronic intestinal symptoms, with alternating periods of exacerbation and remission. Occasionally the disease is ushered in by a fulminant attack with rapid depletion severe toxemia, and often, fatal complications. Similar violent and catastrophic attacks may occur during the exacerbations of a more chronic form The chronic stage may be characterized by a chronic invalidism and inability to resume normal activities Throughout the course of the disease distressing, serious, and even fatal complications may occur These are discussed below

Diagnosis is based on the history of protracted and unusually severe *diarrhea* The stools are *watery* and *bloody* and contain *mucus* and *pus* Frequently pus and blood are passed per rectum without stool These symptoms are associated with abdominal *cramps* and *dyschezia* There is a rapidly progressing anemia and weight loss Physical examination of the patient may reveal a person who is chronically ill with fever toxicity lethargy and weakness or in the case of the *fulminating* type of disease one who is acutely ill only for a period of a few hours with violent diarrhea and blood loss. Fever leukocytosis, toxemia, and anemia are usually present. The disease usually begins in the terminal portion of the colon sigmoidoscopic examination reveals the *hyperemia* and *friability* of the mucosa. Areas of ulceration and hemorrhage with immobility of

the colon, may be observed If the disease has been present very long direct vision of the mucosa may reveal areas of attempted regeneration with characteristic granulation tissue. In the extreme, *pseudopolyp formation* is not infrequently seen Barium study of the colon reveals the spasticity of this organ the loss of haustral markings (lead pipe colon) and if the disease has been going on very long a *shortening* of the entire large bowel, the result of progressive fibrosis (Fig 43)

Differential Diagnosis

Included in the differential diagnosis are other diseases which may be manifested by ulceration of the colon mucosa these include amebic disease bacillary dysentery (and other chronic diarrheas) and enterocolitis *Amebic disease* of the colon is caused by a specific pathogen, *Endamoeba histolytica* Amebic disease although originally tropical in distribution, has in recent years become disseminated and may be found in any part of the civilized world The cecum and rectum appear to be sites of predilection. The diagnosis is usually based on the history of severe and chronic diarrhea with passage of mucus, blood and pus and on the recovery by smear and culture of the specific pathogen Numerous amebacidal drugs are available in the medical armamentarium Surgical therapy of the disease itself is rarely appropriate and is limited to the management of a complication, hepatic abscesses. It is mentioned here largely because of its interest in the differential diagnosis with ulcers in the rectum due to ulcerative colitis The specific pathogen can frequently be recovered from the ulcers on the valves of Houston through the sigmoidoscope. *Amebic typhlitis* enters in the differential diagnosis of granuloma-



FIG. 43 *A* Roentgen ray study of the colon with severe ulcerative colitis. Note furring of the mucosa and "puddling" of the barium. *B* Late stage showing total loss of normal architecture of the colon.

tous disease in the ileocecal area. Amebic abscess of the liver will be discussed in Chapter 33.

Enterocolitis is a disease affecting the small and the large bowel, most commonly in the vicinity of the ileocecal junction. The disease resembles the granulomatous entity *regional enteritis* much more than it does nonspecific ulcerative colitis. There is a marked thickening of the mucosa which resembles granulation tissue. The seromuscular coats are markedly inflamed and edematous. Partial and intermittent obstruction is the rule. Cramps and abdominal tenderness are commonly followed by diarrhea. Systemic manifestations include fever and leukocytosis. Diagnosis is difficult and therapy is the same as if the disease were regional enteritis, i.e., short-circuiting by itself or followed by resection of the terminal ileum and right colon.

Carcinoma of the colon and multiple polyposis of the colon must be included in the differential diagnosis of ulcerative colitis.

Complications

The complications of nonspecific ulcerative colitis are both local (in the colon) and systemic. The local complications include pseudopolyp formation with ultimate malignant degeneration of some of the polyps if total colectomy is not accomplished. Systemic complications are those of arthritis, anemia, and toxemia.

Treatment and Prognosis

Many mild cases of ulcerative colitis have been successfully treated with various biotherapeutic agents, psychotherapy and supportive treatment with blood and other nutritional elements. Acute fulminating cases are still occasionally seen in which no form of therapy is successful and a rapid downhill course leads to early death. If they fail to respond promptly to vigorous medical treatment including the administration of steroid substances, emergency one-stage colectomy may be necessary to prevent death from toxemia, hemorrhage or perforation.

In most cases, however, there is time to carry out a sequential attack upon this disease which has yielded results, which although not perfect, are the best so far attained. A *preliminary ileostomy* is performed so as to divert completely the fecal stream from the diseased colon. Supportive therapy with blood, vitamins, high-caloric intake, and biotherapeutic agents results in marked improvement in general condition. After complete diversion of the fecal stream by ileostomy the hyperemia of the large bowel subsides and the process becomes relatively quiescent. At that time a *total colectomy* is performed. Complete extirpation of the diseased colon removes the cause of toxicity and complications and with the aid of psychotherapy may ensure the patient a long and useful life. Indications for total colectomy are (1) failure to respond to adequate medical therapy, (2) multiple abscess formation including intraabdominal and anorectal abscesses and fistulae, (3) repeated and severe hemorrhage, and (4) the frequently observed complications of (*a*) intractable arthritis, (*b*) cutaneous disturbances, (*c*) regenerative pseudopolyposis, and (*d*) carcinoma developing in an area of regeneration.

When colectomy is performed for ulcerative colitis, the rectum should be removed at the same or a later stage because this segment is generally severely involved. Recently some workers have suggested that the distal 3 or 4 in. of the rectum may be saved and an ileo-terminal proctostomy accomplished with frequent examination of this distal segment endoscopically. Most clinics find that the anastomosis of the ileum to the rectum is almost invariably followed by activation of the disease and carcinoma frequently occurs in the rectal stump even if it is defunctionalized. The last named complication, carcinoma, is assuming increased importance in its relation to ulcerative colitis. The time element is important. If active colitis exists for 8 or more years, the incidence of carcinoma very greatly exceeds the

normal expectancy Furthermore, the malignancy is masked by the colitis, and surgical cure of these secondary malignant neoplasms is virtually unknown

VOLVULUS

Incidence and Etiology

Volvulus is an acute occasionally subacute and not infrequently recurrent accident consisting of a rotation of the intestine about the axis of its mesentery This results in obstruction of the lumen of the alimentary canal, especially if the twist is more than 180° and may be severe enough to compromise the blood supply thus constituting strangulation In infancy volvulus usually affects the small intestine and is associated with malrotation of the intestinal tract or with a very narrow mesenteric root. In the adult, volvulus most frequently involves the sigmoid colon. The anatomic basis of this form of mechanical obstruction is a markedly redundant sigmoid colon on a relatively long mesocolon A similar tendency to volvulus of the cecum is present if there is inadequate fixation of the ascending colon to the parietal peritoneum The greater the mobility of a segment of large bowel, the greater the chances of it twisting upon its mesentery A physiologic predisposition exists in those peoples whose food intake contains a very high residue (such as the Scandinavian and Middle Eastern populations) and who as a result, are subject to chronic constipation The hard stool in the sigmoid colon may eventuate in a violent attempt at hyperperistalsis, which results in twisting

Diagnosis

The diagnosis of volvulus of the sigmoid colon is usually made in an adult who is chronically constipated who perhaps has previously been found to have a markedly redundant sigmoid colon and in whom there is a sudden onset of severe lower abdominal cramps. Enema afford no relief The patient rapidly become violently ill with a silent abdomen fever leukocytosis, and a fast pulse Scout film roentgenography of the abdomen will occasionally reveal the twist of the sigmoid colon into a shape resembling the Greek letter omega (Ω) The air filled loops with the beak at the point where the twist occurs are suggestive of the "ace of spades" Barium enema may confirm these findings by the inability to fill the sigmoid loop beyond the twist. Occasionally sigmoidoscopic examination at this time may reveal the distal end of the twist

Treatment and Prognosis

Treatment of volvulus, as with all potentially strangulating obstructions, requires early relief Occasionally during proctoscopy the cautious passage of a rectal tube through the twist into the distal loop may relieve the obstruction If it does operation can then be performed as an elective procedure, which is safer If the volvulus cannot be relieved by lesser means, immediate operative intervention is mandatory since volvulus produces a closed loop obstruction with all the dangers this implies Simple derotation (detorsion) may suffice although resection of the redundant segment is usually advisable

Volvulus of a mobile cecum is occasionally seen it is more common in the female than in the male Symptoms and signs are localized to the right lower quadrant, and the diagnoses of appendicitis and/or low ileal obstruction are usually made Fixation of the cecum and ascending colon in the right paracolic gutter is usually curative, provided the bowel wall is viable

WOUNDS

Etiology

Wounds of the colon both civilian and military are sustained by a variety of methods and by a multitude of agents and mechanisms Penetrating objects and perforating missiles may enter from any direction on the trunk Wounds of the large bowel are

less frequent than those of the small intestine but, in general are more serious. Associated injuries of other viscera are the rule rather than the exception Blunt or nonpenetrating trauma to the abdomen, such as a direct blow, compression and immersion blast, are not uncommon causes in civilian practice Perforation of the rectum by the insertion of various objects including compressed air are well known particularly in drunk and psychiatric patients.

Diagnosis and Treatment

Surgical exploration, with meticulous examination of the entire coelomic cavity is mandatory whenever an injury to the colon is suspected Initial treatment for shock, of course, takes precedence over the actual celiotomy Small holes in the large bowel may be sutured, with or without proximal decompression. If there is any doubt about the adequacy of the closure or if the wounds are extensive, proximal colostomy or cecostomy must be performed When there is gross destruction of a portion of the wall of the colon particularly of the left colon the damaged segment may be exteriorized to the surface of the abdomen provided it can be done without tension. Retroperitoneal drainage or parasacral drainage of the rectum is frequently advisable

The performance of proximal colostomy for extensive wounds of the colon is highly advisable and the principle bears repetition. Due largely to this operation and to the exteriorization of extensive colon wounds, a tremendous reduction in mortality occurred between World War I and World War II In the former the mortality from large bowel wounds was about 75 per cent, in World War II about 25 per cent. Intestinal antisepsis undoubtedly provided a valuable adjunct, as did parenteral biotherapy and adequate blood replacement

SUMMARY OF PRINCIPLES

1 The large bowel is really two separate organs The right colon is a part of the midgut its main function is to extract water and electrolytes from the stool The blood supply is from the superior mesenteric artery The left colon is a storage and excretory organ it is the hindgut and is supplied by the inferior mesenteric artery

2 The venous and lymphatic drainage of the colon is of clinical importance, since cancer of the colon spreads along these pathways. The lymph nodes are distributed along the course of the arteries that supply the colon. The venous drainage is to the portal system

3 Obstruction of the colon is most frequently due to cancer in its descending portion. Other less common causes are diverticulitis, benign tumors, volvulus intussusception, extrinsic malignant disease, pelvic inflammatory disease, fecal impaction and granulomatous strictures

4 Obstruction of the colon in the presence of a competent ileocecal sphincter results in closed loop obstruction. This must be considered tantamount to a strangulating obstruction because it may result in early perforation somewhere in the obstructed segment or in compromise of its blood supply Immediate operative decompression by cecostomy or colostomy is mandatory

5 In obstruction of the colon, in the presence of an incompetent ileocecal sphincter the differential diagnosis between large bowel obstruction and low small bowel obstruction is difficult because the colon becomes decompressed into the small bowel.

6 The emergency treatment of obstruction of the large bowel is the relief of the obstruction by appropriate decompression definitive treatment of the cause of the obstruction may be postponed until the patient has recovered from the pathologic physiology of the obstruction

7 Polyps of the colon may be pedunculated or sessile the latter should be viewed with much suspicion since there is a high percentage of malignant disease found in them Treatment of a single polyp of the colon is the removal of that polyp and its examination by the pathologist If the pedicle is normal no further operative procedure is necessary If the pedicle or adjacent mucosa are involved with malignant cells and no lymph nodes in the mesentery are palpable segmental resection of that portion of the bowel should be performed If mesenteric lymph nodes are found to be involved with cancer cells a formal extensive cancer operation is indicated

8 Polyposis of the colon is a specific disease characterized by a profound tendency for the mucosa of the large bowel to produce innumerable polyps of every variety and size. One form of familial polyposis is associated with the finding of melanin deposits at the mucocutaneous junctions, particularly of the lips and mouth this is known as *Peutz Jegher syndrome* The treatment of polyposis of the colon is total colectomy

9 Adenocarcinoma of the large bowel is a common alimentary tract cancer Sixty five per cent of all cancers of the colon occur in the rectum rectosigmoid and sigmoid colon The cecum accounts for 13 per cent of all bowel cancers

10 The diagnosis of cancer of the right colon is usually made on the basis of either a progressive anemia of unexplained cause or the palpation of a mass in the right lower quadrant In cancer of the left colon particularly of the sigmoid and rectosigmoid the diagnosis is usually made on the history of passage of blood, a change in bowel habits and the palpation digitally or the visualization endoscopically of a tumor The sequence of examination in all suspected cases is unvarying and includes accurate history digital examination proctosigmoidoscopy with biopsy and roentgen ray examination Exfoliative cytology is valuable

11 The treatment of cancer of the colon is adequate preparation and wide surgical excision

12 Operability and resectability of cancer of the colon in general is much more favorable than in the stomach Five-year nonrecurrence rates vary from 40 to 80 per cent of all cases seen. Longevity is limited by (1) local invasion of adjacent viscera, (2) distant metastases and (3) extensive hepatic involvement

13 Malignant lymphosarcomas of the large bowel are rare. Benign mesenchymal tumors are occasionally encountered

14 Diverticulosis of the large bowel is the presence of outpouchings of mucosa and submucosa at sites of predilection where perforating vessels enter the bowel on the mesenteric side The sigmoid colon is the most common site

15 Inflammatory disease of diverticula diverticulitis may produce spasm of the affected and adjacent bowel intestinal obstruction localized peritonitis, perforation with local or generalized peritonitis and systemic signs of infection

16 Diverticulitis frequently responds well to a medical regimen of low residue diet antispasmodics and biotherapy The indications for surgical therapy are (*a*) obstruction (*b*) recurrent attacks of inflammation (*c*) repeated hemorrhage (*d*) unremitting spasm (*e*) the inability to differentiate this disease from cancer and (*f*) the complications of the disease

17 Complications of diverticulitis are obstruction peridiverticulitis perforation and fistulae between the diverticulitis and adjacent viscera most particularly the urinary tract and the female genitalia

18 Since the most common complication of the operation for resection of the colon in diverticulitis is extension of the infection resectional therapy is usually preceded by proximal decompression the definitive resection follows at an appropriate interval of time

19 Chronic nonspecific ulcerative colitis is a disease of unknown origin characterized by profound inflammatory reaction in the colon and by severe local and generalized symptoms and signs Clinical symptoms include diarrhea, with passage of pus, blood and mucus

20 Prominent in the pathologic findings of ulcerative colitis are chronically inflamed and edematous mucosa and loss of haustral markings in the muscle layers The colon is shortened and assumes a lead pipe appearance

21 Medical management of chronic nonspecific ulcerative colitis is not uniformly successful although some cases become burned out Most often a total colectomy is necessary following defunctionalization of the bowel by a permanent ileostomy Indications for total colectomy are failure to respond to adequate medical therapy multiple abscess and fistula formation the severe complications of intractable arthritis and cutaneous disturbances regenerative pseudopolyposis and carcinoma developing in an area of regeneration

22 Enterocolitis is a granulomatous disease which straddles the junction of the small and large bowel it resembles regional enteritis more than nonspecific colitis. Amebiasis of the cecum is to be remembered in the differential diagnosis of this disease

23 Volvulus of the colon is most common in the sigmoid division and consists of a twisting of the bowel about the axis of its mesentery The resultant obstruction is of the closed loop variety and, unless relieved early will lead to strangulation. The disease is typically sudden in onset and is most frequently seen in persons with a high residue in the colon and in whom there is a markedly redundant sigmoid colon on a long mesentery The roentgenographic Ω sign may be helpful in diagnosis Occasionally the twisted bowel can be derotated with a tube through the proctoscope. Definitive treatment is resection of the redundant colon.

24 Volvulus of a mobile cecum is occasionally seen and is due to the

failure of the cecum and ascending colon to become attached to the lateral parietal peritoneum

25 Wounds of the large bowel are more serious than those of the small intestine because of the difference in their flora. The large bowel is injured by many types of violence to the abdominal wall blunt nonpenetrating trauma as well as penetrating and perforating injuries are common in civilian practice

26 When wounds of the large bowel are suspected, exploratory celiotomy is mandatory Proximal colostomy in extensive injuries to the colon results in a much lower mortality rate than if proximal decompression is not employed. Extensive destruction of large parts of the large bowel may be exteriorized and the colon reconstructed at a later stage.

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Intestinal Obstruction

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DEFINITION AND INTRODUCTION

Intestinal obstruction is an interruption of continuity of the forward passage of intestinal contents from above downward It is a subject of never-ending interest to the surgeon Its ramifications are wide and its facets many Its management requires understanding of many fields in medicine ranging widely from embryology and congenital defects through fluid and electrolyte balance to cardiovascular dynamics and respiratory physiology A case of intestinal obstruction may require for its successful management knowledge gathered from many disciplines as widely separated as biochemistry and hemodynamics, microbiology and cellular physiology pathology and radiology It will become apparent before the end of this chapter that, whereas the surgery of neoplasia and of metabolic errors may well shrink within the foreseeable future intestinal obstruction bids fair to remain a problem for an indefinite time

EMBRYOLOGY AND SURGICAL ANATOMY

Physiologic Division of the Alimentary Tract

The ultimate physiology of the alimentary tract is anticipated in the embryology of the digestive system Early in embryologic development the alimentary tract becomes divided into three separate divisions, each of which will have its own blood supply and perform functions rather sharply delineated from the others Similarly each division of the intestinal tract has individual anatomic characteristics which determine the clinical manifestation of obstruction in that segment

At an early stage in the development of the embryo there is an invagination at the cephalad end and another at the caudad end—the former the *stomatodeum* the latter the *proctodeum* Between these two invaginations the entire alimentary tract, from the mouth to the anus, is developed The detailed development of the alimentary tract into three portions, the *foregut* the *midgut* and the *hindgut* are recorded in standard texts of embryology The important differences in each of these portions with regard to blood supply to organs derived from each division to ultimate function and to obstructive signs in each division are listed in Table 3

Anatomic Division of the Alimentary Tract

The anatomy of the alimentary tract is beautifully *adapted to its several functions* in the processes of taking in nutriment making it useful to the organism and eliminating that part of ingested material which is not useful to the body The function of digestion is served by the foregut. The stomach initiates the process, after mastication and salivary digestion, of breaking down, both mechanically and chemically complex foodstuffs which have been ingested This process continues in the duodenum there other chemical agents and enzymes are brought to the intestine by the organs derived from the foregut, the liver and pancreas, for the purpose of further breaking down complex substances. The celiac axis supplies blood to the foregut and its parenchymatous derivatives.

The function of the midgut *absorption* of digested edible material is carried out by its three segments, each of which has a specific purpose and a corresponding anatomic adaptation. Compared with the ileum the jejunum is thick walled of larger caliber and richer in arterial blood supply and it contains much less fat in its mesentery The ileum on the other hand is thinner walled, with a narrower lumen and a richer supply of veins than of arteries. In the more abun

TABLE 3

ANATOMIC AND PHYSIOLOGIC BASIS IN INTESTINAL OBSTRUCTION

	Foregut	Midgut	Hindgut
Blood supply	Celiac axis	Superior mesenteric artery	Inferior mesenteric artery
Organs	Stomach, duodenum, liver pancreas, spleen	Jejunum ileum, right colon	Left colon
Function	Digestive	Absorptive	Excretory
Obstructive signs	High → vomiting	Intermediate → vomiting and/or distention	Low → distention

dant fat in its mesentery there are many lymphatic channels (lacteals) The differences in anatomy of these two portions of the midgut correspond to the different functions which they serve in the total process of absorption In the jejunum the foodstuffs which have been initially broken down by the foregut are further hydrolyzed by the addition of water and enzymes secreted as succus entericus The large quantity of water poured into the jejunum for this purpose is effected by the lush arterial blood supply of this segment. The adequately hydrolyzed material is then absorbed through the mucosa of the ileum into both the venous and lymphatic circulations. This explains the large number of veins and lacteals in the mesentery of the ileum

Although the large bowel grossly is a single organ and distinct from the small gut with respect to function it is really two organs. The right colon consisting of the cecum ascending colon and right side of the transverse colon, receives its blood supply from the same source as the jejunum and ileum (the other two components of the midgut) the superior mesenteric artery Its function too is *absorptive* indeed it is responsible for the major absorption of water and electrolytes.

The function of the left colon, from mid transverse colon to the anus, is *excretory* Here waste products that are no longer useful to the organism consisting of unusable portions of ingested material excess fluid and enzymes secreted above and desquamated cells of the entire alimentary tract are temporarily stored and then eliminated from the body The blood to this portion of the gut is supplied by the inferior mesenteric artery Since the intestinal content becomes progressively firmer as it is passed downward owing to absorption of liquids, the anatomy of the left colon is adapted accordingly This includes more powerful musculature an appropriate control by the autonomic nervous system, and specific alterations in the mucosa. The terminal colon provides a greater amount of lubrication by the increasing concentration of mucus producing glands in the sigmoid colon and the rectum

These beautiful anatomic and physiologic adaptations of the various portions of the gut are reflected in the clinical findings in intestinal obstruction since the condition varies with the nature of the intestinal contents and the part of the gut obstructed

PATHOLOGIC PHYSIOLOGY

Fluid, Electrolyte, and Acid Base Balance

Until recent years, the morbidity and mortality in intestinal obstruction was very high largely because many factors which were known about the *dynamic physiology* of the alimentary system were not integrated and applied clinically Dynamic, indeed, is the function of a system which uses more water in a day than the entire circulatory system contains at any one time There is a recurring ebb and flow of liquids into and from the intestinal tract, carrying water and enzymes inward and hydrolyzed materials and salts outward saving some things and disposing of others, and at all times maintaining a very important balance with the total circulating volume Interruption of this chain of events at any point may disturb circulatory and acid base balance Basic to the understanding of the pathologic physiology of intestinal obstruction, therefore is an understanding of normal fluid and electrolyte balance described in a previous chapter In addition, however there enter a number of other considerations depending upon the site cause and character of the obstruction to the downward flow of intestinal contents

A list of organs and the daily amounts and general character of fluid produced by each follows

Salivary glands, 200 ml (alkaline)
Stomach, 500 to 1,500 ml (acid)
Bile 500 to 1 500 ml (alkaline)
Pancreas 300 to 1 000 ml (alkaline)
Succus entericus, 1 000 to 5 000 ml (alkaline)

In obstruction high in the intestinal tract vomiting is an early symptom This vomiting carries out an excessive amount of the acid gastric content. This is especially true if the obstruction is at the outlet of the stomach above the entrance of the common duct into the duodenum This excessive loss of the chloride ion disturbs the buffer mechanisms of the blood primarily the bicarbonate and biphosphate systems resulting in a relative excess of base and causing an elevated pH (alkalosis) This is opposite to what happens to the pH of the blood when diarrhea is the condition Then there is a predominant loss of small intestinal contents, which are alkaline in nature under such circumstances there is a relative acidity of the blood with a lowered pH and a reduction of fixed base

In obstruction of the small bowel several liters of various types of fluid may be poured out per day into the lumen of the small intestine which cannot be returned to the circulation in its normal way That portion of *the fluid which cannot be returned is as lost* to the circulation as if it were spilled outside the body for under such circumstances the lumen of the intestine must be regarded as being outside the body Furthermore under conditions of obstruction the fluid cannot be replaced by mouth since the patient usually vomits Such loss of fluid need not occur in a day but may continue over a period of days or even weeks, because intestinal obstruction may be complete partial, or intermittent. The daily loss into the intestinal tract of even a small amount of fluid which cannot be replaced because of a feeling of fullness because of cramps or because of vomiting may gradually lead to a diminished effective circulating volume which is, in effect chronic shock Thus it is clear that the primary cause of morbidity and death in untreated intestinal obstruction is the disturbed physiology of fluid electrolyte and acid base balance This includes the rather special problem of potassium deficits In the healthy potassium is excreted by the kidneys in amounts equivalent to average daily intake that is 50 mEq Potassium loss in the very young whether it is due to diarrhea or to vomiting is particularly serious

When intestinal obstruction occurs low in the alimentary tract as in the distal portions of the small intestine or in the large bowel abdominal distention appears early and regularly The distention occurs because fluid and gas have a large space into which to back up before vomiting occurs This distention is caused not alone by the fluid poured out into the intestinal lumen but also by the accumulation of gases The gases have been shown to be due largely to swallowed air (70 per cent) partly to the diffusion of gases across the intestinal mucosa (20 per cent) and least of all to the putrefactive processes in the intestinal lumen (10 per cent)

Circulatory and Respiratory Complications

Distention of the intestines leads to further *disturbed physiology particularly* of the circulatory system and the blood and also of the respiratory system Dilatation of the intestinal lumen results in stretching and thinning of the bowel wall with compression of its contained vessels particularly the veins, since venous pressure is lower than arterial It is to be remembered that stasis of blood flow is conducive to the development of thrombosis

In addition to distention of the anterior abdominal wall the diaphragm may also be elevated, producing serious respiratory embarrassment. Atelectasis especially of the lower lobes is not uncommon in prolonged abdominal distention in bedridden patients In patients who are elderly and whose respiratory function is already compromised this complication is serious and frequently marginal to recovery The reduced thoracic space due to abdominal distention also increases the load on the right side of the heart and may lead to a fatal outcome before the cause of the intestinal obstruction can be found and relieved

TYPES

Adynamic (Ileus)

Intestinal obstruction may be *adynamic* (neurogenic), *mechanical* or *vascular* Neurogenic paralysis of the intestinal wall results in a failure of the intestine to propel its contents forward This is a form of obstruction however, it is to be sharply differentiated from the other forms described in the following paragraphs Adynamic ileus occurs as a sequel to one of three main causes (1) infection surrounding the intestinal tract such as *peritonitis* (2) mechanical *trauma* including handling of the intestines during a surgical procedure (3) by reflex, disturbances elsewhere in the body such as diaphragmatic pleurisy calculus in the urinary tract and hemorrhage in the retroperitoneal space Obviously the history of any one of the above conditions may lead one to the diagnosis of adynamic ileus, the occurrence of which may even be anticipated Ileus is characterized clinically by diffuse abdominal distention usually associated with vomiting and failure to pass flatus The gravity depends on the underlying cause the intestinal atony itself being well tolerated if not too prolonged Auscultation reveals a *silent abdomen* bowel sounds being absent as a result of inhibition of intestinal peristalsis. Roentgenographically the loops of bowel both small and large are observed to be dilated and paralyzed and occasionally arranged in stepladder formation

Mechanical Simple and Strangulated

Mechanical intestinal obstruction is caused as its name implies by the interposition of something mechanical along the route of the alimentary tract preventing the forward passage of luminal contents This mechanical obstruction may be extraintestinal intramural or intraluminal in origin It may be partial or complete, intermittent or unremitting A mechanical obstruction in which the bowel wall is still viable by virtue of a blood supply which is unimpaired is called a *simple mechanical obstruction* When the blood supply to the intestinal wall is compromised a *strangulated obstruction* exists Thus the difference between a simple mechanical intestinal obstruction and a strangulation obstruction is the *viability* of the intestinal wall dependent upon whether or not the blood supply to the segment involved is compromised Loss of viability leads to the escape of highly virulent microorganisms and toxic products into the peritoneal cavity and constitutes the most frequent cause of death in strangulated intestinal obstruction The second important cause of death in untreated strangulated obstruction is the loss of plasma and blood into the intestinal wall and lumen and into the peritoneal cavity which is severe enough to reduce the effective circulating volume sufficiently to produce profound shock Thus a long strangulated loop produces shock and toxemia more rapidly than a short loop

Vascular

A third type of intestinal obstruction is that in which the cause is of a *vascular* nature. *Mesenteric arterial occlusion thrombosis* or *embolism* rapidly leads to nonviability of the intestinal wall which, from the standpoints of diagnosis and therapy is similar to strangulated obstruction *Mesenteric venous occlusion* is somewhat slower in development but nonetheless carries all the diagnostic and therapeutic implications of strangulated obstruction

When bowel has become strangulated by any of the means here discussed it becomes necrotic if the strangulation is not relieved in time Loss of viability enormously increases the threat to life Removal of the dead tissue is an essential part of treatment. At operation the question of viability or nonviability is not always simple Occasionally the early changes of an impaired circulation can be reversed by removing the obstructing cause and by injecting the root of the mesentery with vasodilators This procedure together with the application of warm saline towels and the administration of 100 per cent oxygen by the anesthetist are sometimes

rewarded by the return of normal color tone and even peristaltic movements to what had been a dusky intestine only a few minutes before If there is any serious doubt about the viability of the gut after these maneuvers or if the gangrene of the intestine is beyond all doubt, resection of the dead tissue is the only treatment that will offer the patient a chance for survival.

SYMPTOMATOLOGY AND DIAGNOSIS

The diagnosis of adynamic ileus rests on the history of some other primary disease since ileus is always secondary Usually there is a history of a very recent operation (within the past 24 to 96 hours) suspicion or evidence of calculus in a duct, such as renal cholecystic, or pancreatic the presence of diffuse or localized peritonitis hemorrhage in the retroperitoneal space whether spontaneous postoperative, or posttraumatic



FIG. 44 Reflex acute gastric dilatation in a patient after an automobile accident with rupture of the liver and injury to the vertebral column

and occasionally diaphragmatic pleurisy or pneumonia, which may also lead to adynamic ileus.

Physical examination characteristically reveals a diffusely distended abdomen which is tender throughout Auscultation will usually reveal reduction or absence of bowel sounds (silent abdomen) from the time of inception of the ileus Roentgenography of the abdomen shows gaseous distention of both small and large bowel, and, if the ileus involves the entire small intestine a stepladder pattern, in which the loops of small intestine appear in parallel layers, one upon another The stomach may be found to be acutely dilated as part of a diffuse ileus or *acute gastric dilatation* may occur as an isolated condition without paralysis of other portions of the alimentary tract (Fig. 44)

The diagnosis of mechanical obstruction is made on the very typical history of *colicky abdominal pain vomiting* and *distention.* The actual site of the obstruction in the intestinal tract will determine which of these three phenomena predominates This has been discussed above One has but to understand the physiology of cramps to appreciate how characteristic is this symptom of a mechanical obstruction When the lumen of a tube which ordinarily possesses peristaltic motion is obstructed there is at first, an attempt to overcome this block by an increased frequency and amplitude of the peristaltic contractions These attempts increase until a maximum is reached, and then the amplitude and frequency of peristaltic contractions rapidly begins to diminish this is due to the exhaustion at the neuromuscular ending a phenomenon characteristic of over stimulation of all biologic tissue The increased amplitude and frequency of the peristaltic rushes" result in intermittent crampy pain they also produce the characteristic auscultatory findings a higher than normal pitch and a greater frequency in the attempt to move the luminal contents forward *The simultaneous occurrence of abdominal cramps and rushes of hyperperistaltic sounds is characteristic of mechanical obstruction*

As time progresses a new type of sound is produced the metallic "tinkling" which is due to peristaltic activity in the presence of an increased amount of fluid and gases

It is seen thus that the physical findings in mechanical obstruction are not static that is they are not the same from hour to hour Rather abdominal auscultation must be repeated very frequently even many times within 1 hr the better to follow the sequence of changes in the bowel sounds and to obtain an impression as to the type and nature of the process causing the intestinal obstruction If prolonged mechanical intestinal obstruction will result in complete exhaustion of the neuromuscular mechanism responsible for the production of peristalsis and thus eventuate in a silent abdomen This sequence and its causes are entirely different from those in adynamic ileus Further examination of the patient with mechanical obstruction will frequently reveal in addition to the auscultatory findings the presence of scars on the abdominal wall due to previous trauma or surgical intervention An external hernia will frequently be found to be present. These signs may suggest the cause and nature of the obstruction

Roentgenography frequently helps to establish the diagnosis of mechanical intestinal obstruction Normally no gas is present in the adult small intestine There is usually gas visible in the small intestine which stops abruptly at the mechanical obstruction and is not seen beyond it. The gas may not be generalized but confined to a single obstructed loop (Fig. 45) There may be one or more loops of small bowel with *air-fluid levels* In mechanical obstruction of the large bowel there may be massive dilatation of the colon down to the point of obstruction distal to which no gas is seen

The differentiation between simple and strangulating obstruction is of paramount importance Strangulated obstruction presents the diagnostic features of a simple mechanical obstruction with certain variations (1) The patient appears more seriously ill, and there are greater departures from general norms the leukocytosis is more exaggerated the fever is higher, the dehydration is greater the shocklike state is more profound and the patient is more toxic (2) There usually is an area palpable on the anterior abdominal wall which is more tender with more *rebound tenderness* than all other areas This exquisitely tender area denotes the localized peritonitis caused by circulatory changes in a segment of intestinal wall (3) Strangulated intestinal obstruction sometimes is diagnosed roentgenographically on the basis of escape of air through the nonviable bowel and the occurrence of pneumoperitoneum

There is one special type of obstruction deserving of special attention This is the *closed loop obstruction* in which a segment of intestine is obstructed at both ends The pathologic significance of this type of obstruction rests on the fact that it cannot be decompressed either proximally or distally Consequently the pressure within it will con-



FIG. 45 Scout film of abdomen showing an isolated loop of small bowel distended with gas. This loop was found, at operation to be strangulated and gangrenous.

tinue to mount until the blood vessels are so compressed that strangulation of the bowel occurs Thus this form of simple obstruction may progress to a stage of strangulation unless early surgical decompression is effected. Clinical examples of closed loop obstruction are volvulus of any loop of bowel and an obstructing carcinoma of the *sigmoid colon in the presence of a compe*tent ileocecal valve

Despite the different manifestations in the various forms of obstruction outlined above it must be realized that there is no infallible means of making a correct differential diagnosis. No one can differentiate between adynamic ileus and mechanical obstruction in all cases an even lower average of accuracy is reached in the differential diagnosis between simple mechanical obstruction and strangulated obstruction Occasionally it will be found that the preoperative diagnostic features and the findings at the time of operation are disparate Nevertheless on the basis of the pathologic physiology the above-listed findings must arouse a high index of suspicion for one or the other types of intestinal obstruction

ETIOLOGY

The causes of mechanical intestinal obstruction will vary in frequency a great deal with the type of practice from which the statistics are culled However by and large there is considerable agreement from many large hospitals and practices with regard to the incidence as expressed in the following

Adhesions (35 to 40 Per Cent)

The peritoneum is a serous mesothelial membrane which responds defensively to all threats or insults from foreign substances or infecting organisms by the reaction of inflammation and exudation Its ability to do this is challenged only by that of the skin Early the inflammatory exudate produces soft plastic *fibrinous adhesions* between various loops of bowel or between the intestines and the abdominal wall These may cause mechanical obstruction of the intestinal tract. Such obstructions may be complete or partial and they may be temporary disappearing when the defensive mechanism of the body has won its battle with the invader This explains the transitory obstructions in the very early postoperative period Later the fibrinous exudate may become organized, that is, may proceed to one of the ultimate end points of the healing process the formation of scar tissue Contrary to widely held popular belief such adhesions are usually not productive of symptoms and are compatible with normal longevity and well-being. Occasionally however they may be responsible for mechanical bowel obstruction The agglutination of intestinal loops to one another or to other viscera or to the abdominal wall may result in kinking or twisting of the intestine of a degree sufficient to obstruct it. More frequently a band of *fibrous tissue* reaching from one area in the peritoneal cavity to another will form under which a loop of small intestine may be caught and completely obstructed If a loop of bowel is so incarcerated it may become strangulated. Under such conditions the course will be progressive and unremitting unless treated surgically

Hernias (35 to 40 Per Cent)

External hernias are by far the most common causes of mechanical obstruction in this group These include inguinal femoral umbilical ventral (early and late) and diaphragmatic varieties In the population at large more inguinal hernias are responsible for intestinal obstruction than any other type because of the overwhelmingly greater frequency of this variety Of a given number of femoral hernias a greater percentage become obstructed and strangulated than of any other type of hernia Umbilical hernias are commonly the site of incarceration of a portion of the omentum with a reflex ileus of the intestinal tract *Internal hernias* including those of the paraduodenal and paracolic sites and those that occur through the

anomalous openings in the mesentery contribute a small number to this group

A special form of hernial strangulation is the so-called *partial enterocele* or *Richter's hernia*. This occurs when a portion of the circumference of the bowel is drawn into a hernial orifice and becomes trapped. This occasionally occurs in the inguinal, femoral and obturator areas. They are important because the mass of intestine in the hernia may be too small to be detected on palpation and further because the entire circumference of the bowel loop is not obstructed and the patient may continue to have bowel movements. These facts make for difficulty in diagnosis.

Malignancy (15 Per Cent)

Most of the malignant tumors of the intestinal tract that cause intestinal obstruction are those of the large bowel, usually of the lower descending colon, sigmoid and rectum. Obstruction of the small intestine by malignancy is usually due to metastases to its serosal surfaces. A very small percentage of obstructions of the small intestine may be due to primary malignancy of the small bowel itself, such as lymphosarcoma, and occasionally to benign tumors. In a practice heavily weighted toward the geriatric side the percentage of obstructions caused by malignancy may well be expected to be higher.

Other (15 Per Cent)

Volvulus. Volvulus is the twisting of a segment of intestine on the axis of its mesentery so as to form a closed loop obstruction. It may occur in the small or large intestine. Volvulus of the small intestine frequently occurs in childhood and is commonly associated with congenital anomalies of fixation and rotation of the intestinal tract. An important anomaly may be hypermobility due to a very narrow mesenteric root. In the large intestine, volvulus usually occurs in the sigmoid colon when this structure is on a long mobile mesentery and particularly in persons or populations subsisting on diets with excessively high residues. This accounts for the geographic variations in incidence throughout the world. Volvulus of the cecum and terminal ileum is favored by incomplete fixation of the ascending colon. Volvulus is particularly apt to occur in the aged because of the tendency to constipation and the redundancy of the sigmoid colon and its mesentery.

Intussusception. Intussusception is the telescoping of one portion of the intestinal tract (the intussusceptum) into another which is immediately distal to it (the intussuscipiens). Common sites are in the small intestine, i.e., ileoileal, or at the junction of the small and the large intestine, i.e., ileocolic, and sometimes a combination of these, ileoileocolic. Intussusception may occur at any age. It is most common in childhood where the story is rather typical of acute bouts of colic with intervening periods during which the child is perfectly well. Frequently in such children a sausage shaped mass can be felt, which is the distal loop of bowel distended by the telescoping into it of the proximal portion. The cause in children is probably intestinal hyperperistalsis. Intussusception in the adult is relatively rare and usually is secondary to some other lesion. This is frequently a tumor of the intestinal wall, either benign or malignant, or some other factor producing hyperperistalsis as a consequence of partial and intermittent mechanical obstruction.

The treatment of intussusception is the same as that of any other obstruction, which may, here as elsewhere, be either simple or strangulated. The removal of the cause of the obstruction is sometimes sufficient to cure the disease, but sometimes the bowel must be resected if nonviability of the intestinal wall has occurred. In infants and small children, barium enemas, which roentgenologically aid in the diagnosis, may also have therapeutic value in reducing the intussusception.

Vascular. Intestinal obstruction on a vascular basis has become more and more common as the average age of the general population has increased. Thromboembolic dis

ease is based on the same causative agents here as elsewhere Mesenteric arterial embolization is an acute abdominal emergency which requires rapid intervention because the gut wall, deprived of its blood supply rapidly becomes necrotic and gangrenous and results in a diffuse peritonitis Mesenteric arterial embolization is consequent upon (1) those diseases in which atrial fibrillation is most common particularly rheumatic fever heart disease with mitral stenosis and (2) left ventricular mural thrombosis consequent upon myocardial infarction.

Spontaneous mesenteric arterial thrombosis occurs just as it does in other relatively small arteries as a sequel to atherosclerosis It has a more insidious onset than the catastrophic ischemia of arterial embolization Mesenteric venous occlusion usually results from some acute infectious process within the abdominal cavity whether a generalized or a localized peritonitis or the infection of a viscus without peritoneal reaction The cause of venous thrombosis in the abdomen is not different from that elsewhere This includes those diseases that predispose to (1) stasis, (2) infection (which produces the bacterial intimal damage) and (3) shortened intravascular clotting time as in the blood dyscrasias and polycythemias primary or secondary

The clinical findings in obstruction on a vascular basis are similar to those of strangulated obstruction therefore this disease deserves the attention and early surgical intervention accorded other types of strangulated obstruction

Regional Enteritis. Regional enteritis (discussed elsewhere *quo vide*) is a granulomatous disease of the small intestine sometimes localized, sometimes diffuse sometimes with a spotty distribution The cramps in regional enteritis are a result of the narrowing of the lumen in the small intestine usually of the ileum and an attempt by hyperperistalsis to force the contents through the narrowed lumen Characteristic of the disease is the formation of adhesions between one loop of small intestine and another Because of the adhesions and the partial intestinal obstruction fistulae may occur between these loops proximal to the site of obstruction The diagnosis of regional enteritis except in its acute stages is usually not difficult The x ray findings are characteristic For the specific considerations in the treatment of this disease entity refer to Chapter 25

Congenital Anomalies Congenital anomalies causing intestinal obstruction in the neonatal period and early weeks of life include the following (1) atresias (complete absence of lumen) (*a*) of the duodenum (*b*) of the distal ileum at the ileocecal area, and (*c*) of the terminal rectum atresias may occur anywhere in the intestinal tract but are most common at the above named sites (2) stenoses (stenosis is the narrowing of the lumen as opposed to an atresia, which means, literally no hole) stenoses are common, in general, in the same areas in which atresias are found, although they may occur at any site in the alimentary tract (3) anomalies of malrotation and/or partial rotation

In the embryologic development of the alimentary tract the intestines are arranged into their final adult pattern by a process of rotation which is described in standard text books of embryology Failure of this process to take its usual course is not completely understood Apparently it may be interrupted at any stage of rotation The right colon may not come to lie on the right side of the abdomen it may be interrupted in its progress toward the right colic gutter or the cecum and ascending colon may end up in the right upper quadrant These are various stages of one and the same process The extreme of such is a reversion down the phylogenetic scale to a condition known as *mesenterium commune* in which the entire intestinal tract arises from a common mesentery in the dorsal midline of the abdomen anterior to the vertebral bodies This anomaly is very likely to result in a volvulus sooner or later although some cases have been seen well along in adult life without

having caused any obstruction The authors have dissected the cadaver of a man past the age of sixty who had no abdominal operations and in whom a mesenterium commune was found High intestinal obstruction may be the result of other congenital anomalies namely (1) the obstruction of the third portion of the duodenum by a very tight superior mesenteric artery as it courses over the third portion of the duodenum after leaving the inferior surface of the pancreas and (2) annular pancreas the obstruction of the second portion of the duodenum by a ring of pancreas arising from the head of that organ The detailed differential diagnoses of these various findings of congenital anomalies are discussed in Chapter 23

TREATMENT

The treatment of intestinal obstruction resolves itself into the three main objectives (1) *reversal of the pathologic physiology* (2) diagnosis and removal of the cause of *obstruction* (3) *the maintenance of general body economy* during the time that the first two measures are being carried out.

Reversal of Pathologic Physiology

It was observed that the basic pathologic physiology is a disturbance of fluid electrolyte, and acid-base balance as a result of the inability of the intestinal tract to handle the ebb and flow of its various fluids, in addition to the abnormal loss of fluids that may occur by vomiting Furthermore the normal physiology is disturbed by the distention causing certain difficulties with the respiratory and circulatory systems

As was observed in Chapter 5 by the time that marked dehydration is recognized clinically the patient has already lost about 6 per cent of his normal body weight. Furthermore one must always be aware of the two main categories in dehydration, i.e., water depletion and salt depletion. Electrolyte deficit may be predominantly on the acid side or on the base side In an attempt to determine a starting point one may use the examination of the urine while awaiting the results of blood chemical investigations Thus employing the Fantus urine test one can determine whether the patient is in relative salt depletion or water depletion A further preliminary opinion will be formed from the history particularly the duration of the obstructive signs the degree of vomiting, and the amount of distention Should one find that the kidneys are excreting less than 3 Gm of sodium chloride per liter he should hasten to replace lost sodium chloride parenterally as soon as possible It must be remembered that one cannot always correct fluid loss and restore normal electrolyte and acid-base patterns in a matter of hours Sometimes it requires as long as several days to accomplish this objective Nevertheless corrective measures should be instituted at once on the basis of initial findings in the urine and later from the blood chemical determinations when laboratory data are available These measures are directed toward restoration of abnormal losses as well as maintenance of current requirements

It is always to be remembered that the amount of fluids the patient can tolerate per unit of time will be determined not alone by what has been lost, but by the ability of his cardiovascular system to accept intravenously administered fluids Thus, several days may have elapsed, and the patient may have lost several liters of fluid per day into the lumen of his intestine and additional quantities by vomiting during which time he has been unable to ingest any fluids thus increasing the deficit. Another source of loss is gastric or intestinal drainage The volume and composition of such fluids must be measured and taken into consideration in computing the replacement program. In addition, the current requirements must also be supplied by parenteral administration Despite the large volumes needed there is a maximum rate at which it can be replaced A planned program is therefore imperative In addition to approximately 5 Gm of sodium chloride per day required for mainte

nance one can replace a portion of the deficit in each 24 hr of treatment The success of such a replacement program will be gauged not alone by repeated laboratory data from urine and blood at frequent intervals but by clinical observation of the patient the degree of dehydration the status of the tension of the eyeballs the wetness of the tongue and the evidence on the skin of dryness In any program of fluid and electrolyte restoration, the degree of success and the time required will be inversely proportional to the duration and severity of the depletion

Not to be forgotten is the fact that the patient needs a minimal daily caloric intake to prevent weight loss during the period of his illness Of particular importance is the question of nitrogen balance In prolonged and debilitating diseases there is a negative nitrogen balance with the depletion of existing body stores of protein, which is reflected by diminished total circulating protein levels In addition it is to be remembered that parenterally administered proteins cannot be utilized as such unless and until the caloric needs have been met. Commercially prepared solutions are now available in a wide variety to meet the different problems presented These include electrolyte solutions which are weighted toward the acid or the base and combinations of protein hydrolysates with caloric providing sugars Throughout the restoration of a relatively normal physiologic state the adequacy of the renal function must be known If it is impaired great care must be taken not to overload the circulation, particularly with respect to the potassium ion Potassium is excreted by the normal kidney in approximately the same amount as the daily oral intake but in renal insufficiency there is a retention of potassium in the circulating blood

In strangulating obstruction, shock due to blood and plasma loss into the intestinal wall and lumen and into the peritoneal cavity demands vigorous attention It is combatted by the judicious intravenous use of whole blood and plasma expanders.

Diagnosis and Removal of Cause of Obstruction

Whatever means are found suitable for the given case *decompression* is imperative This extremely valuable procedure begins the unwinding of the pathologic spiral the removal of accumulated fluids, the prevention of overdistention of the intestinal wall that might threaten its viability the relief of the effects of tympanites on the respiratory system and the avoidance of circulatory complications, such as thromboembolic disease Adequate decompression will also render the gut wall more amenable to safe handling at the time of operation Decompression of a closed loop obstruction is not possible either through the gastric route or per rectum Therefore decompression of a closed loop obstruction when that diagnosis is made, is possible only by an immediate operative procedure Decompression of dilated intestinal tract with all of its untoward sequelae must be accomplished without delay This can be effected in several different ways depending in part, on the level of the obstruction Usually the aspirating tube is placed in the stomach and the contents removed by suction *Gastric* suction alone may suffice but often decompression of the small intestine directly by means of an intestinal tube is more effective Certainly the closer to the obstructed site one can place the tip of the aspirating tube, the more decompression at the critical point will be obtained and a number of intestinal tubes have been designed for this purpose There are however some objections to their use First, it is mechanically difficult for the tube to pass beyond the pyloric sphincter and through the duodenum It tends to become kinked or rolled back upon itself in the stomach The difficulty is increased by the absence of normal peristalsis When normal peristalsis is present a tube passes easily because of the fact that the peristaltic motions pucker the intestine on the tube (a condition known as the pleating effect)

Surgical Therapy

Definitive therapy in mechanical obstruction is operative correction of the cause of the occlusion. Proper timing of the operation is a most important factor in the outcome of the disease. No operation is required for adynamic ileus per se. However, the condition causing the ileus, such as peritonitis, often demands operative therapy. In simple mechanical obstruction there is almost always sufficient time to restore the patient's fluid, electrolyte, acid-base and nitrogen patterns to or toward normal before operative therapy is undertaken. This may require a few hours or a day; in the extreme 2 days may be required. When the diagnosis of strangulated intestinal obstruction is made the definitive therapy is operative correction *now* with a minimum of time loss for preoperative preparation. Thus, whereas intestinal decompression may be definitive for adynamic ileus, it is preoperative therapy for simple mechanical obstruction and not to be too prolonged preoperatively for strangulated intestinal obstruction. Closed loop mechanical intestinal obstruction is to be treated as a strangulated obstruction. In mechanical intestinal obstruction decompressive therapy must be looked upon as a preoperative measure only, its duration to depend upon the type of obstruction. It should not be depended upon as definitive therapy for the obstruction. The objectives of operation are to release the obstruction by whatever means are suitable and to remove nonviable segments of intestine and restore continuity. The manner of handling dead (nonviable) bowel has been discussed earlier in this chapter.

Maintenance of General Body Economy

Throughout the patient's illness, from the onset of symptoms until he is restored to health, his general body economy must be maintained by means other than natural. The alimentary tract is the ideal route for intake of all materials used to maintain nutrition and provide for daily repair of metabolized tissue. When it cannot so be used, all attempts to maintain norms must be through the parenteral route. In its broadest meaning, parenteral alimentation must include not only restoration of disturbed fluid and electrolyte requirements, but maintenance of a normal blood profile through the use of whole blood and blood substitutes. The protein content of circulating blood is but a reflection of protein stores, which must be kept at relatively normal values with these agents. Calories, vitamins, and antibiotics must be judiciously administered in optimal quantities via the parenteral route when indicated. Through adequate pre- and postoperative care the patient will best be able to withstand and benefit from definitive surgical therapy.

SUMMARY OF PRINCIPLES

1. The diagnosis and therapy of intestinal obstruction is based on the pathologic physiology of the disease.

2. The basic pathologic physiology of intestinal obstruction is the disturbance in fluid, electrolyte and acid base balance and the toxemia resulting from dead bowel and infected peritoneum.

3. In strangulating obstructions loss of plasma and blood into the bowel wall and lumen and into the abdominal cavity may cause shock from reduced effective circulatory volume.

4. Serious sequelae to the entire organism may result if this pathologic physiology is not rapidly reversed.

5. Complications of the pathologic physiology include serious respiratory and cardiovascular embarrassment.

6 There are several types of intestinal obstruction (*a*) adynamic ileus intestinal paralysis, secondary or reflex to other disease (*b*) simple mechanical obstruction intestinal obstruction without compromise of intestinal blood supply (*c*) strangulated obstruction including closed loop obstruction, intestinal obstruction with compromise of blood supply to the bowel wall proceeding to nonviability and necrosis if not corrected

7 The main clinical causes of intestinal obstruction are adhesions hernias malignancies congenital anomalies volvulus intussusception, vascular diseases and regional enteritis.

8 The treatment of intestinal obstruction involves the restoration of the disturbed physiology including fluid and electrolyte imbalance and shock intestinal decompression and operative removal of the obstruction with a properly timed surgical intervention In strangulated obstruction where the pathologic physiology is most severe surgical intervention must be early and is directed toward the removal of nonviable bowel. Attention must be meticulous to maintain adequate nutrition normal hemogram positive nitrogen balance and cardiovascular renal homeostasis

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29

Peritonitis

I Anatomy, physiology, and repair of the peritoneum
II Causes of peritonitis
A Bacterial
B Chemical
C Neoplastic
III Types of peritonitis
A Localized
B Generalized
IV Pathologic physiology
A Large infected surface
B Ileus
V Diagnosis
VI. Sequelae
A Ileus
B Adhesions and fistulae
C Toxicity
VII Treatment of peritonitis
A Treatment of cause
B Treatment of ileus
C Treatment of infection

ANATOMY, PHYSIOLOGY, AND REPAIR OF THE PERITONEUM

The peritoneum is a vast membranous sheet one cell layer in thickness which covers all the intraabdominal viscera and lines the coelomic cavity It performs several functions (1) It prevents friction between viscera which change position in the course of their normal function Thus, changes in position and size of the various loops of bowel the bladder and a growing uterus slide smoothly upon both the visceral and parietal layers of the peritoneum permitting accommodation to various functional states (2) It absorbs both fluid and particulate matter into the blood and lymph streams the rapidity of absorption of injected materials from the peritoneal cavity is second only to the intravenous route Particulate matter both microorganisms and inanimate material accidentally or traumatically introduced is engulfed by macrophages and then absorbed (3) The role of the peritoneum in defense against infection is pertinent to the subject of peritonitis it is equaled only by the skin in its capacity to cope with invading organisms Infection introduced into the peritoneal cavity excites an inflammatory exudate by which the effort is made to localize the process If the defensive effort is successful, the infection may be overcome completely Lesser degrees of defense usually result in walling off the peritonitis,

with the formation of an abscess In the peritoneal cavity the wall of the abscess may *differ from that of a soft tissue variety in* that it may be composed largely of loops of intestine the thickened peritoneum and the greater omentum which descends via some chemotactic attraction to assist in walling off the infectious process It is possible that antibodies are formed in the peritoneum as they are in the skin. Fibrinous adhesions and fibrous scars are early and late evidence, respectively of such defensive action

CAUSES OF PERITONITIS

The causes of peritonitis may be bacterial chemical neoplastic, or traumatic the latter includes the aseptic peritonitis of handling viscera, particularly with cotton sponges.

Bacterial

The most common cause of the peritoneal inflammatory process is *bacterial invasion* of the peritoneal cavity Most frequent sources of bacterial contamination are contents which have escaped from the alimentary tract The healthy gastrointestinal mucosa is impermeable to the microbiologic flora which is indigenous to it When however the integrity of the intestinal wall is breached infection of the previously sterile surface results This breaching may be in the form of actual perforation or by increased permeability of a severely damaged or inflamed intestinal wall (*Durchwandering*) Familiar examples of such invasion are perforation of an inflamed appendix or of a peptic ulcer strangulated intestinal obstruction (seepage) strangulated intestinal obstruction with necrosis of the bowel and trauma to hollow viscera whether penetrating, perforating or blunt. The so-called *aseptic peritonitis* occasionally observed is probably due to unrecognized microorganisms such as viruses

Chemical

Chemical peritonitis is due to a chemical irritant and may occur in the absence of bacterial infection *Hemoperitoneum* blood in the peritoneal cavity is the most common example The sources of such blood include traumatic rupture of solid viscera, such as the liver and spleen tearing of mesenteric vessels by blunt or penetrating injuries or by sudden deceleration in gravitational trauma bleeding ovarian cysts and ectopic pregnancies Another cause of chemical peritonitis is the presence of bile in the peritoneal cavity either as a result of trauma or perforation due to disease or following surgical intervention *Bile peritonitis* occurs without bacterial superimposition but it is a far more serious disease when there is secondary bacterial contamination. Another relatively common cause of chemical peritonitis is hydrochloric acid in the early stages following perforated peptic ulcer Owing to the highly acid content of the stomach the resulting peritonitis is usually relatively sterile for a short period of time If it persists longer bacterial superimposition is the rule

Neoplastic

A third form of peritonitis is *neoplastic Carcinomatosis peritonei* is the name given to the studding of the peritoneal surfaces by metastatic malignancy The malignant implants in themselves are irritant to the peritoneum, and an exudation usually follows.

TYPES OF PERITONITIS

Localized

When the peritoneal cavity is exposed to the presence of a foreign and irritating material whatever its nature (chemical bacterial neoplastic) it rises to the occasion and mobilizes its defenses. It causes adhesions of other peritoneal surfaces in an attempt to localize the peritonitis. It brings in blood cellular elements. It produces an inflammatory exudate it walls off the infection If successful it results in the production of a localized peritonitis with or without abscess

If the insult to the peritoneum is so overwhelming that the peritoneum is unable to limit the spread of the infection the entire peritoneal surface in all its reaches and recesses, becomes involved The signs and symptoms then are those of *generalized peritonitis* which if bacterial becomes an acute suppurative peritonitis it may also be chemical or neoplastic. Many factors are involved in determining to what extent peritonitis can be localized Among the variants are the virulence of the infection versus the resistance of the host and the time element, the rapidity and duration of contamination The concept of resistance has always been rather nebulous and difficult of definition There is some recent evidence that resistance may soon be definable in terms of actual production and circulation of specific chemicals such as the protein *properdin* Tuberculous peritonitis is rarer usually chronic and of an exudative nature It is more common in those areas of the world where infection with *Mycobacterium tuberculosis* is present

PATHOLOGIC PHYSIOLOGY

Large Infected Surface

The disturbed physiology of peritonitis revolves about the problem of infection in a very large space and the paralysis of the intestinal tract which follows closely upon it. The tremendous absorptive expanse of the peritoneum absorbs liquids and particulate matter very rapidly Extremely rapid absorption of infectious material from the peritoneum quickly leads to signs and symptoms of sepsis and the severe toxemia that characterize the disease The rapidity of onset of toxemia and sepsis will depend on whether the peritonitis is relatively localized or is diffuse and generalized

Ileus

The paralysis of the alimentary tract (ileus) leads to disturbances in physiology which follow intestinal obstruction from any cause including those of fluid and electrolyte balance and the circulatory and respiratory complications This is discussed in Chapter 28

DIAGNOSIS

The diagnosis of peritonitis is based on the *local abdominal findings* of infection which may be localized to one part of the peritoneum or extended throughout the entire abdominal cavity and the *systemic manifestations* of infection In addition, the *ileus* which is consequent upon the peritonitis produces all the findings that are characteristic of it.

Since peritonitis in not a primary disease one must seek to identify the disease which may be antecedent to it The list of possibilities includes all the so-called *acute surgical conditions of the abdomen* Any disease which may lead to the perforation of a viscus and the contamination of the peritoneum with intraluminal contents must be considered Any organ which, when inflamed may lead to an exudate on its surface may provide the source The presence of irritating substances not normally in the peritoneal cavity may also be the cause of peritonitis these include free blood, bile, or pancreatic juice All of these materials irritate the peritoneum and cause a spasm of the anterior abdominal musculature muscle guarding which is mediated through the intercostal nerves which innervate the anterior abdominal wall Tenderness is elicited Rebound tenderness which is pain produced by the sudden release of the anterior abdominal wall after manual compression, is due to the friction of the inflamed visceral and parietal peritoneal surfaces Tenderness direct or rebound, may be found diffusely throughout the abdomen in cases of generalized peritonitis in a restricted area in cases of localized peritonitis These findings may change from hour to hour or even from minute to minute since a previously well localized peritonitis

may become diffuse and spreading and may then give generalized findings In generalized peritonitis there will be an ileus of the entire intestinal tract and a progressive distention of the entire abdomen Vomiting usually is present early in the course of the ileus The systemic findings include those of generalized infection, to wit, fever leukocytosis with a high polymorphonuclear cell count and tachycardia. Respirations may be increased in frequency if distention is severe Although it is not always possible to establish the cause of the peritonitis when this diagnosis is made nonetheless one must seek it, since it may determine the plan of therapy

SEQUELAE

Ileus

The earliest sequel to generalized peritonitis is ileus This is a protective mechanism and results in the immobilization of the intestine which favors localization However it is not an unmixed blessing, since fluid and electrolyte imbalance follows upon it. Respiratory and circulatory complications may occur later

Adhesions and Fistulae

When the peritonitis is purulent in character adhesions between and among the loops of bowel particularly the small bowel may form rapidly (depending upon the antecedent disease) Such adhesions may lead to intestinal obstruction and abet the formation of fistulae from one loop of bowel to another

In the process of the subsidence of peritonitis with or without operative intervention the fibrinous adhesions may become devascularized with time into fibrous bands or sheets. This is perhaps the most common cause of intestinal obstruction

Toxicity

The absorption of toxic material from pus, infected bile or infected blood from the tremendous peritoneal surface rapidly leads to a deterioration of the patient's entire body economy The patient appears "toxic." This toxicity is manifested by an increased hemolysis of red blood cells, peripheral vascular collapse marked muscular weakness and septicemia.

TREATMENT OF PERITONITIS

Treatment of Cause

In the treatment of peritonitis there are three main objectives first, the *disease which caused the peritonitis* must be treated second, the *ileus* which has untoward sequelae of its own should be combatted third, the *generalized infection* must be treated The treatment of the cause of the peritonitis is of paramount importance and must not be delayed If the peritonitis is due to and maintained by a hole in or seepage from the intestinal tract, the continuing leak must be shut off or the body's defenses will not be able to overcome the continued contamination The peritoneum is usually able to cope with a single bacterial insult but prolonged reinfection overtaxes its defensive capacities

Treatment of Ileus

The treatment of the ileus in peritonitis should be instituted as soon as the diagnosis is made or even when it is suspected It implies, essentially intestinal decompression from above with gastric or intestinal tubes and restoration of fluid and electrolyte balance to or toward normal The treatment of the infection also is started as soon as peritonitis is suspected or as soon as the systemic signs and symptoms of the peritonitis become manifest. This treatment today involves mainly the use of proper antibiotics by the parenteral route intravenously or intramuscularly The maintenance of the general body economy in the postoperative course is an essential feature in the total management This is described in detail in Chapters 5 and 12.

Treatment of Infection

In the treatment of the peritonitis by operative intervention the subject of *drainage* of the peritoneal cavity has evoked great discussion. Surely it is possible to drain a localized abscess which has been walled off, i.e., delimited from the rest of the peritoneal cavity. The localized accumulation of pus and debris can be drained to the outside by appropriate tubes. However, in a generalized peritonitis it is better to close the abdomen without drainage. It has been repeatedly shown that it is really impossible to drain the general peritoneal cavity. The peritoneum quickly forms pockets and recesses which are inaccessible and the drainage tube becomes walled off. As a foreign body it impairs the ability of the peritoneum to control the infection. Once the source of contamination has been eliminated and the gross quantities of infected material aspirated during the operation, the general body defenses will usually overcome the remaining infection. These defenses are aided by antibiotic-containing fluid and phagocytic blood elements mobilized to the peritoneal cavity. Drainage of the abdominal wall down to the peritoneal level may be indicated if it has been grossly contaminated.

SUMMARY OF PRINCIPLES

1. The peritoneum has a greater surface area than the skin.
2. The defensive abilities of the peritoneum are rivaled only by the skin.
3. Peritonitis is almost always secondary to some disease or trauma.
4. Bacterial peritonitis is usually due to contamination of peritoneal surfaces by intraluminal contents of the alimentary tract.
5. Chemical peritonitis includes hemoperitoneum, biliperitoneum, pancreatic juice and gastric juice peritonitis, and carcinomatosis peritonei.
6. Depending on bacterial virulence and resistance of defense mechanisms and on the nature of the underlying disease, peritonitis may be geographically localized or generalized.
7. The essential pathologic physiology of peritonitis includes (*a*) a massive infection on a vast absorptive surface and (*b*) the sequelae of a paralyzed bowel.
8. The diagnosis of peritonitis is based on (*a*) evidence of peritoneal irritation (pain, tenderness, rebound tenderness), (*b*) the manifestations of ileus (distention, vomiting, fluid and electrolyte imbalance, respiratory and circulatory complications), (*c*) the manifestations of a severe systemic infection (fever, leukocytosis, tachycardia, hemolysis, sepsis, and toxemia).
9. The treatment of peritonitis is directed against (*a*) the underlying cause, (*b*) massive infection, and (*c*) the complications of the paralyzed bowel.

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30

The Rectum and Anus (the Anorectum)

I Surgical anatomy and physiology
II Anomalies of the rectum and anus (see Chapter 23)
III Benign tumors of the rectum
- A Polyps
- B Mesenchymal tumors

IV Carcinoma of the rectum
- A Etiology and incidence
- B Pathology
- C Diagnosis and differential diagnosis
- D Treatment and prognosis

V Inflammatory diseases of the rectum
- A Lymphogranuloma venereum
- B Other granulomas

VI Procidentia of the rectum
- A Etiology and types
- B Treatment

VII Injuries of the rectum
- A Types (1) violence (2) irradiation
- B Treatment

VIII Hemorrhoids
- A Incidence and etiology
- B Diagnosis
- C Treatment
- D Complications (1) stricture (2) pectenosis

IX Cryptitis, fissure, fistula, and abscess
- A Etiology and pathologic types
- B Diagnosis and differential diagnosis
- C Treatment
- D Differential diagnosis of anal and rectal fistulae
- E Complications

SURGICAL ANATOMY AND PHYSIOLOGY

The rectum is the terminal portion of the alimentary tract and consists of two portions, the pelvic rectum (10 to 15 cm in length) and the perineal rectum or anorectum (4 cm in length) The rectum begins at about *the level of the third sacral vertebral body* and lies partially within the pelvic cavity and partially retroperitoneally That portion which is retroperitoneal (the terminal 10 cm) has no serosal cover The rectal cavity is narrowest at its beginning and at its end in between which there is the rectal ampulla usually divided into segments by well-de

veloped rectal valves (Houston) The rectum is maintained in its position by the peritoneum its vascular attachments and the muscular levators In the pelvis the peritoneum is reflected anteriorly onto the uterus in the female and the bladder in the male forming a distal pouch of peritoneum known as the *cul-de sac of Douglas* The vascular attachments of the rectum are (1) the superior hemorrhoidal artery which is the terminus of the inferior mesenteric artery and supplies the largest part of the rectum (2) the two middle hemorrhoidal arteries, which are branches derived from the hypogastric artery they supply the lower mid rectum and (3) the two inferior hemorrhoidal arteries branches of the internal pudendal arteries which supply the anal canal from the pectenate line distally to and including the anus The venous drainage of the rectum corresponds to its arterial supply This drainage is of interest and importance in that the superior rectal circulation is thus tributary to the portal venous system and that of the lower rectum to the caval system The significance of this relationship will be discussed later

The lymphatic supply of the rectum parallels the course of the blood vessels which supply and drain it. This too is of great importance in therapy and prognosis, as will be brought out clearly later in this chapter The significance lies in the fact that malignant metastases from the superior rectum follow the course of the veins which are tributary to the portal system, but those from the midrectum follow the course of the hypogastric arteries to the deep iliac nodes and those from the anal canal follow along the internal pudendal artery to the inguinal lymph nodes

The nerve supply to the rectum is from the autonomic nervous system the parasympathetic division (sacral) is motor to the rectum the sympathetic supply (lumbar) is inhibitory

The rectum lies in intimate relationship with other pelvic viscera and structures It is suspended and held in position by the levator ani muscles which are in reality a sling of muscles which suspend to the bony pelvic brim all the viscera that pass through the pelvic outlet Dorsal to the rectum lies the relatively mobile coccyx, from which the rectum is suspended by a small muscular sheet of the coccygeus muscle In the operation of coccygectomy this close relationship must be borne in mind Because of the relatively superficial position of the rectum at this point a sacrococcygeal approach to tumors of the lower rectum is occasionally employed On its ventral surface the rectum is in close relationship to the nerves arising from the hypogastric plexus, which innervate the bladder In resections of the rectum injury to these nerves may occur with consequent bladder dysfunction In the female pelvis the ventral wall of the rectum is in intimate relationship with the sacrouterine ligaments and the posterior vaginal wall. The rectovaginal septum in multiparous women may be no more than two layers of mucosa, one vaginal the other rectal In the male Denonvillier s fascia separates the ventral wall of the rectum from the prostatic capsule The seminal vesicles especially if inflamed and enlarged, are palpable through the rectal mucosa just cephalad to the prostate

The mucosa of the rectum is richly endowed with mucus-producing glands, composed of high columnar epithelium At the anocutaneous junction there is a transition from epidermoid cells through a narrow band of cuboidal epithelium to the typical rectal mucosal pattern Everted mucosa, however may become so metaplastic that its epithelium resembles epidermis rather than mucous membrane.

The anal canal or terminal 4 cm of the alimentary tract, is guarded by two sphincters the larger internal and the smaller and more distal external The upper limit of this segment, the anorectal line is sometimes referred to as *Hilton s white line* although in reality it is blue to purple The lower border of the internal sphincter is marked on the mucosa by a fibrous ring on the

mucosal surface known as the *band of pecten* or the *dentate line* It is of surgical anatomic importance since it marks the dividing line between the blood supply from the middle hemorrhoidal vessels superiorly and the inferior hemorrhoidal vessels distally Just proximal to the pectenate line are the folds and crypts of Morgagni These folds contain the terminal branches of the veins which drain the anorectum and, under conditions to be described later in this chapter become internal hemorrhoids The crypts are the recesses between the Morgagni folds. When irritated the base of these crypts can hypertrophy with a ring of triangular shaped papillae (papillitis or cryptitis) (Fig 46)

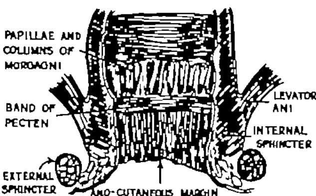


FIG 46. Anatomy of the anorectum

The rectum is the final receptacle for waste products which have traversed and accumulated in the alimentary tract before they are expelled The process of expulsion defecation is a complex neuromuscular action The impulse to evacuate the rectum is initiated by distention of this structure the afferent stimuli ascending the autonomic nervous system pathways Usually the emptying of the rectum is then effected by the action of sacral parasympathetic-controlled longitudinal smooth muscles of the rectum with the assistance of the accessory use of skeletal muscles, under voluntary control The autonomic and automatic part of the reflex consists in contraction of the longitudinal musculature of the rectum together with relaxation of the sphincters particularly the larger internal Expulsive force is voluntarily added by contracting the abdominal musculature and increasing the intraperitoneal pressure by straining with the glottis closed Emptying of the rectum is then followed by relaxation of the muscles which have just contracted, and contraction of the *sphincters which have just relaxed. Pain in* the anal area occasioned by inflammatory or other disease there may interfere with relaxation of the sphincters and thus with the process of defecation. Stricture of the distal rectum will result in a distention of the rectum and rectosigmoid colon Disturbance of the reflex control of the anal sphincters is referred to as *dyschezia*

ANOMALIES OF THE RECTUM AND ANUS

Anomalies in this area are discussed in detail in Chapter 23

BENIGN TUMORS OF THE RECTUM

Polyps

Adenomatous polyps frequently occur in the rectum and are often found to be the cause of bright red rectal bleeding They are visible through the proctoscope and should be removed through this instrument When pedunculated, they may be generously excised including a segment of mucosa at the base and examined histologically Sessile polyps must be viewed with *suspicion and* removed with a generous area of surrounding normal appearing mucosa Polyps are important because they are generally believed to be precursors of carcinoma. For this reason, detection and removal are an important means of cancer prevention in this susceptible area. Electrocoagulation of these lesions is sometimes practiced

Mesenchymal Tumors

Mesenchymal tumors of the rectum are occasionally seen Benign connective tissue tumors in the muscular wall are occasionally seen as are also submucous lipomas Endo-

metrioma may penetrate the rectal wall and cause bleeding from the rectum. It is suspected when a woman of menstruating age bleeds from the rectum with each period.

CARCINOMA OF THE RECTUM

Etiology and Incidence

The rectum, anus, and rectosigmoid junction is the second most common site for cancer in the alimentary tract (stomach rating first). Sixty five per cent of all cancers of the colon occur in the regions mentioned. The age incidence parallels that of cancer anywhere, being most common between the ages of forty and seventy; however, no age is immune, and these lesions are found not infrequently in the very young. This frequency is based, perhaps, on the large number of mucus producing glands. The cause of carcinoma here is not known, but there are at least three predisposing factors in cancer of the anorectum. They are (1) the tremendous increase in the number of mucus-producing glands in this area, for purpose of lubrication, (2) the distinct propensity for metaplasia of mucosa at the always irritated anocutaneous junction, and (3) the distinctly high incidence of excrescences of the rectal mucosa into polyp formation.

Pathology

Most carcinomas of the rectum are adenocarcinomas and are annular in nature; the lateral feature accounts for the early evidence of obstruction. These lesions frequently are ulcerating in character and account for rectal bleeding. Tumors of the anocutaneous junction are usually epidermoid.

Cancer of the rectum spreads by several methods according to its position (and according to the biology of cancer, about which very little is known). As we noted above, cancer of the rectal ampulla and rectosigmoid junction usually metastasizes along the inferior mesenteric artery and vein and thence to the paraaortic lymph nodes and to the liver. Cancer of the lower rectum, which is supplied by the middle hemorrhoidal and inferior hemorrhoidal vessels, spreads to the deep iliac nodes and to the inguinal areas. It usually infiltrates locally early and becomes fixed to the pelvic walls. Cancer of the anal canal spreads very rapidly to the inguinal lymph nodes. A malignant lesion in the area of the pelvic diaphragm usually extends early laterally through the fascia propria and the levator ani muscles to involve the urogenital apparatus. Remote metastases, of course, are always possible and are not infrequently seen; they are due to hematogenous spread. The wide dissemination of these tumors is accounted for by the presence of cancer cells in circulating blood. The growing ability to detect these cells in peripheral blood may make for earlier diagnoses in the future. This is an extremely interesting and important avenue of research at the present, with important diagnostic and therapeutic implications.

Diagnosis and Differential Diagnosis

The diagnosis of cancer of the rectum is made on a fairly characteristic story and an unvarying sequence of examination. The story most commonly is that bright blood is present at or on the stool or is expelled from the rectum by itself. Change in bowel habits toward increasing constipation is characteristic. The patient may have been constipated for many years, but he now reports that the symptom is so severe that enemas are repeatedly resorted to. Before long obstipation (failure to relieve constipation with enemas) is reported. The stool frequently is reported as being pencil sized in diameter. When the tumor ulcerates, diarrhea may occur. Not infrequently diarrhea alternating with constipation is reported. With continuing obstruction abdominal distention is noted to be progressive, and sacral and flank pain are not uncommon.

Digital examination of the rectum is of paramount importance in the diagnosis of

rectal carcinoma. Indeed it *must* be a part of *every* physical examination, since it may detect lesions which have not yet caused sufficient symptoms to bring them to the attention of the patient. The palpation of a mass or indurated ulcer or the presence of bloody material on the examining glove immediately alerts one to the probable presence of a lesion which must be considered to be malignant until proved otherwise

The second indispensable part of the examination is the visualization through the proctoscope Proctosigmoidoscopy too should be a part of routine examinations. In the properly prepared bowel the entire anus and rectum to its junction with the sigmoid can be clearly visualized and if lesions lie at a higher level, the presence of bloody material coming down from above is seen. Biopsy of any visualized lesion or suspected area, for histologic examination, is a part of this endoscopic procedure Study of exfoliated cells from the rectum (in the absence of visualization of a lesion) has yielded a high percentage of positive diagnoses in malignant disease of this structure

Examination of the abdomen may reveal the presence of a pelvic tumor and, very late of a nodular metastasis to the liver and/or ascites Early however none of these findings will be present Pain cachexia, and weight loss are very late symptoms, as are involvement of structures of adjacent systems, such as the urogenital apparatus

The differential diagnosis of cancer of the terminal bowel includes all granulomas and strictures. Histologic examination after appropriate biopsy is of critical diagnostic value

Treatment and Prognosis

Treatment of malignant disease of the anorectum is almost exclusively surgical Surgical therapy consists in wide excision of the tumor-bearing viscus with all of its accessible lymp node–bearing area, usually determined by the main arterial blood supply of the segment involved Thus, ideally the inferior mesenteric artery should be ligated at its origin from the aorta, and all bowel which it supplies (the left colon, sigmoid rectum, and anus) should be resected. In addition the loose areolar tissue around the rectum, and whatever lateral extension that has occurred, should be removed if feasible· this may include adjacent viscera of other systems requiring the sacrifice of internal genitalia. Removal of the anorectum necessitates artificial stomas for excretory function. A portion of bowel wall distal to the lesion at least 5 cm should be resected with the tumor since it has been shown that retrograde extension along lymph channels is common to this extent If the removal of 5 or 6 cm of rectum distal to the lesion leaves sufficient rectal wall to which a portion of the descending or transverse colon can be anastomosed, continuity is restored in this manner If sacrifice of this short segment of distal rectum brings one near the anus where lymphatic drainage would carry the disease to the inguinal area, a combined abdominoperineal resection (Miles) with sacrifice of the sphincters and anus, is indicated for best results. A permanent terminal colostomy is provided

Resectability of rectal lesions has become increasingly high, being considerably better than that of the stomach, for example In rectal tumors resectability probably is as high as 80 per cent of all cases seen. Prognosis for survival best expressed as 5 year nonrecurrence rates, depends on several factors The histologic classification of the lesion, according to either Broders or Dukes is important. It has clearly been shown in many large series that the best survival is obtained with the lowest grade of malignancy and that metastatic lymph node involvement is found to increase rapidly and extensively with grades III and IV and much later and to less extent with grades I and II Where no lymph nodes are found involved at the time of the initial resection 5 year nonrecurrence rates are as high as 80 per cent The over all 5-year nonrecurrence rates for all tumors in this area are about 60 per cent

Lymphosarcoma of the rectum far less common than carcinoma in this area has the same prognosis as it does elsewhere in the alimentary tract in general these lesions are radiosensitive but, unfortunately, hematogenous spread may occur early

INFLAMMATORY DISEASES OF THE RECTUM

Lymphogranuloma Venereum

The most frequently found cause of inflammatory rectal stricture is lymphogranuloma venereum which is caused by a specific virus. The disease is usually not primary in the rectum but extends to the rectum by lymphatics surrounding the genital apparatus This occurs primarily in females, because of the difference in lymphatic drainage from the external genitalia Preputial infections drain toward iliac lymph nodes vaginal drainage is through the lymphatics in the rectovaginal septum Healing of the active lesions results in heavy scar formation and cicatricial stenosis of the rectal lumen Diagnosis is made conclusive by the Frei test. There is no specific treatment for the disease The stricture especially when advanced, responds little if at all to dilatation Multiple fistulae from the diseased rectum onto the perineum and buttocks are common Permanent colostomy with or without resection of the stenosed rectum may become indicated

Other Granulomas

Other granulomatous diseases of the rectum which present symptoms of rectal bleeding, pain, and possibly stricture formation must be considered in the differential diagnosis of all symptomatic rectal disease Amebiasis is not infrequently discovered by the typical ulceration of the rectal mucosa and the isolation of the specific pathogen from these lesions Gonorrhea is occasionally seen in the lower third of the rectum and microscopic and bacteriologic identification is made by biopsy Tuberculous disease of the anorectum is rarely identified by histologic and bacteriologic examination of material taken especially in the presence of fistula formation

PROCIDENTIA OF THE RECTUM

Etiology and Types

Procidentia of the rectum is a disease in which part or all of the rectum prolapses through the retaining sphincters First degree prolapse may consist only in the expulsion from the rectum of mucosa A complete prolapse is the extrusion through the anal canal of the entire wall of the rectum which becomes doubled upon itself It is probably helpful to think of a complete procidentia of the rectum as an intussusception of the rectum through the anus The cause of prolapse or procidentia, of the rectum is varied and usually consists of more than one factor Although occasionally seen in infants it is most common in the aged Chronic severe constipation with marked straining at stool is usually elicited in the history of these patients Additionally the patient is usually of the type who has inherited connective tissue which has little supportive strength These patients usually have varicose veins, hemorrhoids, visceroptosis, and pendulous breasts

Treatment

Treatment of procidentia of the rectum is initially conservative with an attempt to replace the intussuscepted (or "extussuscepted") colon This is best accomplished in the knee-chest position but force should not be used, lest the colon wall become edematous and strangulated Definitive therapy is surgical Corrective procedures, in general, have not been uniformly successful, but the abdominal approach has given best results This consists of mobilization of the rectum and fixation to the lateral pelvic fascia If there is a marked redundancy of the sigmoid colon this segment of the large bowel should be removed with end to-end

anastomosis The reefing operation from below occasionally suffices for less severe cases Amputation from below is fraught with danger frequently leading to damage of a trapped segment of bowel with ensuing peritonitis In the correction of this disease one must assiduously avoid creating an annular stricture

INJURIES OF THE RECTUM

Types and Treatment

The rectum is injured by perforating and penetrating missiles much the same as other segments of the alimentary tract When a bullet or other high velocity missile perforates the rectum there is almost always associated damage to the urogenital apparatus and frequently to the parasympathetic sacral nerve supply to pelvic viscera. Blast injuries and the insertion of foreign bodies into the rectum not uncommonly result in perforation of the rectum into the free peritoneal cavity Blunt trauma to the pelvis as commonly seen in automobile accidents frequently damages the rectum along with the bladder and the pelvic bones Damage to any one of these structures demands examination of all pelvic viscera Exploratory laparotomy is probably safest when a rectal injury is suspected Parasacral drainage of rectal wounds below the peritoneal reflection and proximal colostomy for wounds above the peritoneal reflection are indicated.

Injury to the rectum is frequently seen as a sequel to therapeutic roentgen irradiation for malignant disease of the cervix uteri and, less commonly for osseous pelvic pathology This is similarly true for the insertion of radium needles into the cervix Late injury *occurs in the form of stricture of the rectum* Such a cicatrix does not respond favorably to dilatation and must sometimes be relieved by a permanent colostomy with or without resection of the stenosed area

HEMORRHOIDS

Hemorrhoids are dilated veins of the hemorrhoidal plexuses, of which there are two The *superior hemorrhoidal plexus* consists of those submucosal veins which drain into the middle hemorrhoidal veins, and the *inferior hemorrhoidal plexus* consists of those submucosal and subcutaneous veins which drain into the inferior hemorrhoidal

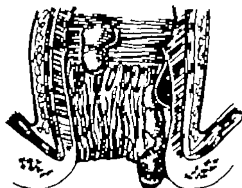


FIG. 47 Internal and external hemorrhoids.

veins The former are called *internal hemorrhoids* the latter *external hemorrhoids* The former are covered by mucosa, the latter by skin More frequently than not they coexist (Fig 47)

Incidence and Etiology

Hemorrhoids are an extremely common human affliction They occur at any age but more commonly in middle and later life and there is little sex difference excluding the increased incidence during pregnancy In many cases no cause of hemorrhoids is to be found These occur in persons who have an inherited predisposition which consists of a tendency toward *laxity of connective tissue* structures these persons are inclined to have varicose veins certain types of hernia, hemorrhoids, lax abdominal musculature and sometimes visceroptosis *Increased hydrostatic pressure* in the superior middle and inferior hemorrhoidal veins (which have no valves) is considered a predisposing factor Such a situation obtains when there is portal hypertension due to tumors or thromboses or when cirrhosis of the liver is present obstructing the outflow of blood from that organ Repeatedly raised intraabdominal pressure also increases the hydrostatic pressure in the terminal venous

plexuses around the anorectum and is conducive to the formation of hemorrhoids These include *chronic constipation pregnancy chronic cough* and *prostatic obstruction* of the urethra Clothing or appliances which constrict the abdominal cavity exert a similar effect

Diagnosis

Both internal and external hemorrhoids may coexist and be entirely asymptomatic A frequent presenting symptom is bright blood seen at the time of passage of stool This is due to bleeding from one of the dilated veins the wall of which has been breached after the mucosal covering has been ulcerated or torn Frequently the patient complains of pruritus and soiling in the perianal area. These symptoms are caused by the escape of rectal secretions which make for moisture and maceration of the skin Frequently the internal hemorrhoid may be so large and the dilated veins so loose upon the submucosa that they are prolapsed through the anal sphincters during the act of defecation This may be painful and may be associated with free bleeding as the mucosa becomes excoriated and ulcerated and in the extreme the protruded hemorrhoid may become edematous and strangulated It cannot then return within the anal canal and becomes a situation demanding urgent care More frequently the blood in these dilated veins becomes thrombosed and inflamed, resulting in acute pain with the sensation of a mass at the anus which consists of the thrombosed vein and the edema surrounding it

The diagnosis is confirmed by inspection of the anal area, by digital examination of the anal canal, and by visualization of this area with the anoscope All stages of hemorrhoids are thus visualized, the acutely thrombosed hemorrhoid, the ulcerated internal hemorrhoid, the protruding hemorrhoid, and, not infrequently the strangulating hemorrhoid. The anastomoses between the superior and inferior hemorrhoidal plexuses is one pathway for collateral circulation in portal vein obstructions Hemorrhoidal plexus dilatations for the purpose of providing such pathways are therefore physiologic compensations and must not be mistaken for primary hemorrhoids

It is worthy of emphasis that the diagnosis of rectal bleeding must never be assumed to be hemorrhoidal in origin until full investigation of the rectum and rectosigmoid has been made by sigmoidoscopic examination and roentgenographic study of the lower colon This is imperative because of the extreme importance of not overlooking malignant disease in the terminal area of the bowel even if obvious hemorrhoidal disease is seen

Treatment and Complications

The therapy of hemorrhoids varies with the stage of the disease Presence of asymptomatic hemorrhoids, if no ulceration is visualized on anoscopy does not call for treatment. The treatment of an acutely thrombosed hemorrhoidal varix consists of incision into the thrombosed area with extrusion of the clot This is a minor procedure accomplished in the office and offers gratifying relief from the acute discomfort. The presence of large internal hemorrhoids which are ulcerated or which protrude with or without the association with external hemorrhoids, constitutes the main indication for formal surgical hemorrhoidectomy Some surgeons still occasionally inject sclerosing solutions into the submucosa to shrink the hemorrhoidal varices Although there may be a useful place for this procedure much better results, in general are obtained by formal hemorrhoidectomy This consists in the meticulous excision of the plexus of veins both internal and external (superior and inferior) with their overlying mucosa and skin. There usually are three or four such groups of dilated veins When care is taken to avoid injury to the sphincters and to allow a strip of mucosa to remain between each excised bundle of veins results are good and complications few Anocutaneous line stricture may result if too much

of the anocutaneous margin is cut away and if circular rather than radial incisions are made at the level of each varix Such an occurrence is apt to cause permanent and distressing symptoms from a "buttonhole" anal aperture

CRYPTITIS, FISSURE, FISTULA, AND ABSCESS

Etiology and Pathologic Types

These four diseases are different stages in a complex series of events which practically always begins in an anal crypt Cryptitis, or papillitis an inflammatory response to a small tear in the anal crypt probably due to passage of hard stool or some foreign body in the stool is very common There is hypertrophy of the anal papillae in response to breach of the mucosa in the anal crypts Whether the papillitis is found with or without hemorrhoids it should be pointed out that one should not be too vigorous in excising too much tissue at the base of the columns of *Morgagni* lest the pectenate line immediately distal to it become scarred occasionally resulting in a very disturbing stricture at this area, referred to as *pectenosis* If the mucosal tear fails to heal and becomes

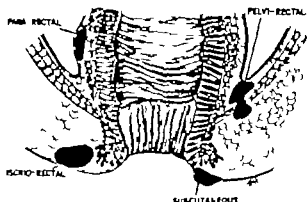


FIG 48 Location of abscesses associated with sinus-fistula disease The subcutaneous abscess is external to the external sphincter the ischiorectal is in the space so named between the skin and the levator ani muscles If the abscess is above the levators, it is pelvirectal dissection of the abscess between rectal wall and peritoneum makes it pararectal

indolent, it is referred to as an *anal fis* or *ulcer* When a perforation occurs f this anal ulcer through the rectal wall ei above or below the levator ani muscle *rectal* or *anal sinus is said to exist.* As burrowing sinus traverses the soft tis around the rectum the bacterial flora of rectum may produce an abscess, whic outside the rectal wall These abscesses n be either in the ischiorectal space (*ischio tal abscess*) or in the pelvirectal space (*virectal abscess*) or in the pararectal sp (*pararectal abscess*) (Fig. 48) Refere to the diagram reveals that the ischiore and pelvirectal spaces are separated by levator ani muscles the pelvirectal and pararectal spaces are separated by the fa propria of the rectum When the abs erupts onto the skin, creating a double-en passage connecting the rectum and the ternal surface of the skin a *fistula* ex (fistula is derived from the Latin w meaning pipe, with two openings) (F 49)

Anorectal fistulae have a *primary op ing* the original start of the disease on inside of the anal canal and *second openings* on the surface of the skin arou the anus The secondary openings onto surface may be multiple Most fissures a therefore the initial penetration of the *tulae occur in the dorsal half of the* circu ference of the anus, although fistulae in ventral half of the circumference are n unusual. Openings around the anus with 2 cm of the anus usually empty radia directly into the anal canal Openings wh are beyond 2 cm from the anus almost ways empty into a primary opening whi is in the posterior or dorsal one-half of t anal canal In order to do this the pathw between the primary and secondary ope *ings is frequently circuitous.* This set of c cumstances follows *Goodsall's rule*

Diagnosis and Differential Diagnosis

The diagnosis of anal fissure is usually n difficult. Severe boring anal pain whi makes defecation an excruciating ordeal

almost always due to fissure The rich sensory innervation of the perianal area plus the *sphincter spasm* triggered by the inflamed fissure explain the severity of the discomfort. In addition, there is often a small amount of blood noticed on the cleansing paper On inspection one sees a *sentinel pile* a response to the local inflammation There is marked sphincter spasm which resents the examining finger because of the local pain produced When the patient feels a mass in the anal area, it presages the formation of a perianal ischiorectal or pelvirectal abscess from a burrowing sinus. Not infrequently the patient will complain that a spot of pus appears on the clothing or that purulent material is extruded with the stool Such symptomatology is suspicious of the existence of a fistula. The patient may complain only of pain at the anus aggravated at the time of defecation but with no drainage This is certainly suspicious of an anal ulcer Systemic signs of infection including fever and leukocytosis may accompany fistulous disease with or without abscess

Examination is first directed to inspection of the anal and perianal area An asymmetry may be noticed with a hot tender swollen area to one side of the anal aperture Digital examination will confirm the presence of a mass which may be superficial to the external sphincter may be deep to it but external to the levator ani muscles (ischiorectal) or may be deep and high, above the levators (pelvirectal) Inspection may reveal a draining opening near or far from the anocutaneous line Digital exam-

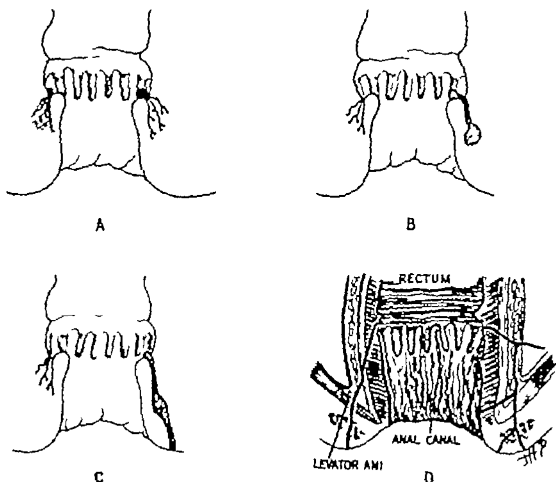


FIG. 49 Pathogenesis of fissure-sinus-abscess-fistula disease of the anorectum *A* A fissure has begun in a crypt between the columns of Morgagni *B* It has burrowed through the wall and is a sinus now The infection may balloon out into an abscess. *C* It has now burrowed its way through the soft tissues and erupted upon the skin. It is now a fistula. *D* A variety of fistulous pathways

ination may reveal a fibrotic tract extending to or toward the primary mucosal opening Anoscopy may reveal this primary aperture but it is best visualized under anesthesia. Although it is possible gently to probe fis tulae and sinuses such examination is best accomplished when anesthesia is adequate

The differential diagnosis of these dis eases includes all diseases of the anorectal canal including neoplastic, inflammatory and granulomatous

Treatment

Anal fissure or anal ulcer may occasion ally be treated conservatively with emollient substances or by cautery to produce fibrosis with subsequent healing If resistant to this type of office procedure, the fissure or ulcer must be excised in toto including its base and the papilla proximal and the sentinel pile distal to it. Partial division of the ex ternal sphincter at its posterior decussation is helpful in relieving spasm associated with *the disease and its treatment.*

Acute abscesses of the ischiorectal space must be opened under anesthesia, as indi cated in the diagram with breaking up of the loculations in the ischiorectal space If the abscess is in the pelvirectal space inci sion must be carried through the levator ani muscles and adequate drainage assured After the abscess cavity has become com pletely obliterated whatever remains of the sinus or fistulous disease can then be treated in sequence

Fistulous tracts between the anorectum and the external skin must be dissected free and entirely excised This is a limited pro- cedure if the fistulous tract is on either side of the external sphincter However if the fistulous tract is deep to the internal sphinc ter and high in the anal canal the dissection may be very extensive It becomes necessary to incise the sphincters of the anus The external sphincter should be incised only at one point at any one time and this pref erably in the posterior line where its fibers decussate When the internal sphincter is divided it must be reconstructed since it is the main guardian of continc tracts of the fistula are further exa the insertion of a flexible probe a sibly by visualization with hydrog ide discolored with methylene blu the fistulae on the external surface ary openings) are found to be mul correction of these complicated r tulae should be done in stages. In stage the ancillary openings should nected with the main secondary op the outside of the body When all tracts have been converted into o another operation is performed to the outside or secondary opening primary or internal opening, as d above Not infrequently the ancilla nal openings are so numerous and sc cally infected, with multiple absces no local procedure will be rewardin the rectum is first defunctionalize colostomy for a period of many When the infection has subsided t *lous disease can be treated from* bel after complete healing in and aro anus and rectum the colostomy closed Fistulae that are not too d have only one secondary opening on may be treated by a simpler amb method A *silk string* or *seton* is with the aid of a probe through the f tract into the rectum and then brou through the anal canal and the tw tied snugly This tight ligature w through the fistulous tract at a ra enough to permit healing At interv seton is tied more and more snugly cuts through to the exterior leaving a tract behind it.

Differential Diagnosis of Anal and Rectal Fistulae

All lesions around the anus and pe which may be associated with either a or drainage must be differentiated fro fistulae These include all tumors in th granulomatous disease (particularly ly granuloma venereum) ulcerative coli lonidal sinuses, presacral dermoids,

bursitis suppurative lymphadenitis infections of the urethral (Littre) glands and osteomyelitis of the sacrum or coccyx

Complications

The main complication of fistulous disease of the anus and rectum is chronicity of infection If not attended early and adequately chronic suppuration of the entire perineum and even of the buttocks may result despite a defunctionalizing colostomy Involvement of the external genitalia in the female and the membranous urethra and prostate in the male is common Rectal incontinence may result from the presence of extensive fistulous disease and not infrequently from the surgical treatment thereof Extensive reconstruction of the guardians of continence is occasionally necessary

SUMMARY OF PRINCIPLES

1 The rectum is the terminal portion of the alimentary tract and is anatomically and physiologically well adapted for temporary storage and the final excretion of alimentary wastes

2 The venous and lymphatic drainage of the rectum and anus are of extreme importance in treatment and prognosis of cancer in this area

3 The upper rectum is drained to the portal venous system the anorectal canal to the caval system The lymphatic drainage of the upper rectum is along the paraaortic lymph nodes that of the lower rectum and anus to the iliac and inguinal nodes

4 The rectum is adapted to the handling of solid material by the concentration of a great number of mucus producing glands This is probably of some etiologic significance in the large number of cancers of this portion of the bowel

5 Anal continence is due largely to the stronger superior or internal, sphincter and to less extent to the more distal and superficial external sphincter

6 See Chapter 23 for a discussion of anomalies in this area

7 Polyps of the rectum are common They must be removed for examination since they may be precursors of carcinoma Sessile polyps must be removed with an area of apparent normal mucosa surrounding the polyp for histologic examination

8 The area of the rectum anus and rectosigmoid junction is the second most common site for cancer in the alimentary tract Carcinomas of the rectum are adenocarcinomas those of the anocutaneous junction are epidermoid

9 Cancer of the rectum spreads by direct extension to the pelvic walls along lymphatic pathways and by the hematogenous route

10 Cancer of the rectum is suspected whenever there is bleeding per rectum a history of progressive constipation obstipation alternating diarrhea and constipation or progressive abdominal distention

11 The examination for cancer of the rectum, or for any disease in this area, follows an unvarying sequence This includes (*a*) detailed history (*b*) digital examination of the anus and rectum (*c*) endoscopic examina

tion with the proctosigmoidoscope and biopsy of any suspicious areas for histologic examination and (*d*) roentgenographic examination

12 Treatment of malignant disease of the anorectum is almost exclusively surgical Operations are designed to remove the diseased bowel and as much of the lymph node–bearing area to this segment as is possible This may occasionally involve associated viscera of other systems. When the lesion is at or below the pelvic peritoneal reflection this almost always implies an abdominoperineal resection (Miles) with permanent colostomy

13 Resectability of rectal and sigmoidal lesions is high, approximating 80 per cent 5 year nonrecurrence rate is similarly high approximating 50 to 60 per cent.

14 Lymphosarcomas of the rectum are usually radiosensitive but may have already spread by the hematogenous route

15 Inflammatory diseases of the rectum particularly lymphogranuloma *venereum frequently result in rectal stricture, which does not yield to dila*tation

16 Procidentia of the rectum is essentially a herniation of rectum through the anal sphincters Many degrees are encountered the more severe in the aged and surgical correction by abdominal operation is usually necessary

17 Injuries to the rectum by external violence are not uncommon in industrial and automotive accidents and they frequently require proximal colostomy The rectum may be injured by roentgen irradiation for malignant disease of the cervix uteri and frequently may require proximal colostomy

18 Hemorrhoids are dilated veins of the hemorrhoidal plexuses are common at all ages, but are predisposed to in patients with loose connective tissue with severe and protracted constipation or with portal hypertension or chronically raised hydrostatic pressure due to multiple pregnancies or abdominal tumors Hemorrhoids may be the source of bleeding as well as of much pain due to acute thromboses they may become ulcerated may protrude through the anal canal and may occasionally become strangulated

19 Asymptomatic hemorrhoids need not be treated surgically Acute thromboses may be incised and drained and large, protruding or ulcerated hemorrhoids should be excised by meticulous dissection

20 Even when hemorrhoids are found on anal examination bright rectal bleeding must not be assumed to be due to this disease without complete examination of the lower colon

21 Cryptitis anal fissure anal fistula and abscesses in the anorectal area are four stages of a complex series of events occurring at the anorectal line

22. Cryptitis or papillitis is an inflammatory disease indicating a defensive mechanism against infection or trauma Anal ulcer or fissure is most common at the dorsum of the anus

23 A burrowing sinus may result in abscesses in the area which are known according to the space involved these may be either ischiorectal pelvirectal or pararectal

24 Anal fissure is best treated by excision of the ulcer together with its associated papilla and sentinel pile Acute abscesses must be opened under general anesthesia and loculations broken up

25 Fistulae may be multiple and together with their associated abscesses may produce severe and chronic infections in the perineum and buttocks

26 Extensive fistulae of the anus and rectum are best treated surgically in stages by complete excision with reconstruction of the muscular sphincters Simple fistulae may be treated on an ambulatory basis by the seton treatment

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The Management of Gastrointestinal Hemorrhage

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INTRODUCTION

Gross bleeding from the gastrointestinal tract is a frequent occurrence often perplexing as to cause and sometimes most difficult to control It may require treatment before the cause is determined or the location established The severity of the hemorrhage varies widely from acute and massive and perhaps quickly fatal, to minor episodes of repeated bleeding The blood may escape from the mouth by vomiting or be expelled from the rectum In general bleeding high in the alimentary tract (including esophagus stomach, and duodenum) will result in the vomiting of blood However it is possible that hemorrhage may originate in any of these sites without vomiting and the blood proceed down the alimentary tract at varying speeds. When blood travels through the alimentary tract slowly it is decomposed and presents in the rectum as a tarry substance giving the stool a characteristic coal-black appearance *melena.* Hyperperistalsis induced by the irritative presence of blood in the intestinal lumen causes such rapid transit that blood extravasated high in the intestinal tract may appear in the rectum while still bright red. Blood shed into the lumen of the colon almost always appears in the rectum while still bright. Anal bleeding characteristically appears with bowel movements and the blood is not intimately mixed with the stool

UPPER TRACT

Differential Diagnosis

Peptic ulcer is a very common disease and is the principal cause of bleeding from the upper intestinal tract. It must always be considered even when there is no antecedent history of the disease since not infrequently

gross hemorrhage is the first sign that an ulcer exists Occasionally one may elicit the recent history of aggravation of the signs of underlying peptic disease (burning pain empty feeling and the taste of blood) *Cancer* of the stomach is the second most common cause of upper intestinal bleeding In a given number of gastric carcinomas a larger percentage bleed than do in a similar number of cases of peptic disease however carcinoma of the stomach is not as common in the general population as is peptic ulcer Hemorrhage from *bleeding esophageal varicose veins* is the third most common cause of massive upper alimentary tract hemorrhage and is found in patients with hepatic cirrhosis (when the blood cannot get out of the liver) and in extrahepatic portal hypertension (when the blood cannot get into the liver) Under these conditions the esophageal veins form an important collateral pathway for returning portal blood to the caval circulation Evidence that these diseases have pre-existed is frequently available in the form of a history of cirrhosis or the palpability of a grossly enlarged liver Other evidence may exist in the form of enlarged collateral veins of the abdominal wall and ascites Benign lesions of the stomach including *polyps* and *hypertrophic gastritis* must also be considered as causes of high bleeding Other causes present in the duodenum and upper jejunum although less frequent, must also be included in the differential diagnosis These include tumors of the papilla of Vater and of the extrahepatic biliary ducts and benign tumors of the duodenum and proximal jejunum.

Symptomatology and Signs

When bleeding from any of these high lesions occurs slowly the blood will be digested in the intestinal tract and the symptom that brings the patient for medical care is melena, tarry black stool It has been estimated that the presence of 60 to 100 ml of blood in the intestinal tract is required before the stool is turned black On the other hand, if the bleeding is massive bright blood will usually be vomited (*hematemesis*) In the former case laboratory examination may reveal evidence of chronic blood loss in the blood profile since bleeding has been slow enough to allow for dilution When bleeding is sudden and massive there is just one outstanding symptom and sign *shock* which is recognized by weakness pallor ashen gray color and diaphoresis These are all the result of *acute hypovolemia* More often than not there will be no local signs

Treatment

The therapy of shock takes precedence over the treatment or even the diagnosis of the underlying disease As detailed in Chapter 4 this means primarily the replacement of whole blood early and in adequate amounts The efficacy of the replacement of lost blood is assessed by repeated laboratory determinations of circulating blood volume and the blood profile If the suspicion of bleeding esophageal varices is strong on the basis of collateral information available by history or observation, control of the shock includes an attempt to prevent further hemorrhage from the submucosal veins of the lower esophagus by the placing of a balloon (Sengstaken) in an attempt to compress the bleeding varices and thus prevent further loss of blood This apparatus consists of a tube to which are attached two balloons one to engage the cardia of the stomach and the other to compress the submucosal veins of the esophagus By aspiration separately of stomach and esophagus, the site of bleeding may be determined If the index of suspicion is sufficiently high attempts may be made to visualize the dilated veins radiographically with the aid of a barium swallow even though the patient is critically ill Any risk thereby added in the patient's management is compensated for when a positive diagnosis is made In general, x ray examination of the esophagus, stomach, and duodenum is now being made earlier than was previously practiced As soon as shock has been overcome and bleeding from the esophagus is controlled immediate steps are taken to estab-

lish a diagnosis so that definitive therapy may be instituted Use should be made of all available laboratory methods of exam ination *in an attempt to pinpoint the diagnosis* as early as possible In the definitive man agement of massive bleeding the surgeon is always faced with the choice of immediate operative versus delayed therapy In peptic disease emergency surgery is demanded un der two main conditions The first condition obtains if adequate blood replacement fails to restore normal vascular dynamics within a reasonable period of time (12 to 24 hr) It is worthy of repetition that blood replacement must be early and adequate and that pulse and blood pressure must remain normal after the transfusions have been discontinued If the blood pressure falls and the pulse rises with evidence of a diminished blood volume, operation must be resorted to if the mortality rate is to be kept at a mini mum Repeated lapses into shock may produce irreversible changes in parenchymatous organs under these conditions there is no alternative but immediate operation The second condition obtains if bleeding recurs while the patient is still under observation

In general in known peptic disease the consensus is that conservative therapy may be followed without great risk to the patient when (1) the current episode of bleeding is the first (2) the patient is less than forty years of age and (3) response to the early hours of conservative therapy is gratifying by maintenance of normal cardiovascular dynamics with a minimum of blood replace ments Militating for early operative therapy are (1) previous episodes of bleeding (2) a patient over forty years of age (3) diffi culty in maintaining normal cardiovascular dynamics without repeated blood transfu sions, and (4) evidence of continued bleeding In the case of esophageal varices either failure to stop the bleeding with the com pressive balloon or recurrence of the bleed ing when the tube is removed requires im mediate operative intervention After the epi sode of acute hemorrhage is over and the patient's condition is improved definitive therapy of these conditions must be carried out according to indications detailed in other chapters

LOWER TRACT

Differential Diagnosis

The most common cause for blood pre senting from the rectum, i.e at the anus is *local lesions in the anorectal canal* These include *bleeding hemorrhoidal varices fis sures* and *fistulae* Bleeding from these sources although consisting of bright blood is rarely of massive proportions Certainly examination of this area, both digitally and with a good light and scope must precede all else The most common serious lesion in the lower intestinal tract which produces bleeding per rectum is *cancer of the rectum and rectosigmoid colon* In every case of rectal bleeding this lesion must be considered and must be sought for This includes *digital examination* and *visualization* per sigmoidoscope of the distal 25 cm of the alimentary tract. Cancer of the colon above the sigmoid colon is also a frequent contributor to the finding of blood per rectum *Diverticulitis* of the left colon particularly of the sigmoid, is a disease of great fre quency and is a common cause of bleeding *Benign tumors* of the colon, especially *mucosal polyps* can be productive of massive bleeding Occasionally such a polyp is torn from its pedicle by a piece of hard stool resulting in massive hemorrhage *Multiple polyposis* of the colon has a high incidence of serious bleeding this diagnosis is usually known to the patient because of previous episodes of bleeding Rectal bleeding, usually not of a severe nature is not an infrequent accompaniment to *strangulated bowel* This is particularly true of strangulation of the sigmoid due to volvulus and of intussusception Laceration of the intestinal mucosa at any level by an ingested *foreign body* has been known to produce bleeding of serious proportions *Other causes of blood appear* ing at the anus, as has been stated, may

actually be found high in the intestinal tract These may include bleeding from peptic dis ease of the duodenum or of the stomach in the presence of hyperperistalsis and a rapid transit through the alimentary tract Osler Weber Rendu disease congenital and diffuse *hemangiomatosis* may be the cause of repeated massive gastrointestinal hemorrhage and because of the nature of the mucosal lesions may defy diagnosis in life and indeed even at autopsy Solitary hemangiomas also cause repeated hemor rhage beginning at an early age in life

In infants and children the most frequent cause of red blood escaping from the rectum is peptic ulceration in Meckel s diverticulum The hemorrhage is due to peptic changes in and around the diverticulum as a result of heterotopic gastric mucosa Although most common in infancy it may occur at any age

Symptomatology and Signs

The systemic manifestations and signs of massive lower intestinal bleeding are no dif ferent from those of massive bleeding of the upper intestinal tract (or for that matter from any source) These include the recog nizable picture of shock, although lower in testinal bleeding less often produces pro found shock than upper alimentary tract bleeding The local symptomatology is the finding of either melena or gross bright bleeding per rectum The two may coexist. It is even possible that tarry stool may be found when the bleeding is very low in the intestinal tract This occurs when there is retrograde flow of blood from the distal into the proximal colon and in unusual circum stances (with a patent ileocecal sphincter) even into the small intestine The blood may be retained so long that when it is finally expelled it has been decomposed and pre sents as melena Such cases have been de scribed following operations upon the anus in which there is a spasm of the sphincters with long term retention of blood Procto scopic examination during a period of active bleeding is sometimes very helpful in local izing the site and source of the hemorrhage

Treatment

The treatment of shock as elsewhere takes precedence over all local therapy Ex amination of the anus and rectum digitally may be followed when the lower bowel may safely be cleansed by proctoscopic examina tion When active bleeding has subsided the colon may be visualized roentgenographi cally provided such sick patients are gently handled Due care must be exercised not to overlook sources higher in the intestinal tract as the cause of blood presenting per rectum. After shock has been controlled and pre liminary examinations have been made fur ther definitive diagnosis for all possible le sions may be made as detailed in the con sideration of each specific lesion in previous chapters

SUMMARY OF PRINCIPLES

1 Massive gastrointestinal hemorrhage requires emergency measures of treatment even before the cause of hemorrhage has been determined

2 The therapy of shock takes precedence over the treatment or even the diagnosis of the underlying disease Early and adequate restoration of normal circulating blood volume, preferably by compatible whole blood is of the greatest importance

3 Massive gastrointestinal hemorrhage may be manifested by the vomiting of bright blood (hematemesis) the passage of decomposed blood per rectum (melena) or the passage of bright blood per rectum

4 The most common causes of upper intestinal bleeding are (*a*) pep-

tic disease (*b*) gastric cancer (*c*) esophageal varices and (*d*) benign tumors of the stomach and gastritis

5 In hemorrhage due to peptic disease emergency surgery is demanded (*a*) if adequate blood replacement fails to restore normal vascular dynamics within a reasonable period of time (12 to 24 hr) and (*b*) if bleeding recurs while the patient is still under observation

6 Militating for conservative therapy of the bleeding peptic ulcer are the following factors (*a*) the current episode of bleeding is the first (*b*) the patient is less than forty years of age (*c*) response to early hours of conservative therapy is gratifying by maintenance of normal cardiovascular dynamics with a minimum of blood replacement

7 Operative therapy in hemorrhage from peptic disease is indicated when (*a*) vital signs are not maintained after the initial correction of the hypovolemia, (*b*) the patient is over the age of forty (*c*) obvious arteriosclerosis and/or hypertension are present and (*d*) there is history of previous hemorrhage

8 Esophageal varices may be suspected if there is a history of intrahepatic disease extrahepatic portal obstruction ascites or an enlarged liver

9 Evidence for esophageal varices may be sought roentgenographically even while the patient is bleeding

10 The Sengstaken tube may be used to compress temporarily bleeding submucosal esophageal veins

11 Common causes of bleeding from the lower intestinal tract are (*a*) local lesions of the anorectal canal (*b*) cancer of the rectum sigmoid, and higher colon (*c*) diverticulitis (*d*) benign tumors of the colon (*e*) strangulated bowel (*f*) Meckel s diverticulum and (*g*) bleeding from lesions higher in the alimentary tract

12 The source of bleeding from the lower bowel must be sought for by digital examination endoscopic examination roentgenographic study and the consideration of sources of bleeding from higher in the alimentary tract

SUGGESTED READINGS

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The Extrahepatic Biliary System, The Metabolism of Bile; Jaundice

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BASIC CONSIDERATIONS

Surgical Anatomy

The extrahepatic biliary passages are a collecting, storage and conduit system for the conduction of bile from the liver to the duodenum. They consist of the main hepatic ducts (usually two) a common hepatic duct, the gallbladder and cystic duct, and the common bile duct The gallbladder is a thin walled sac with a capacity averaging 1 to 2 oz, usually lying imbedded in a fossa on the inferior surface of the liver and covered by a layer of peritoneum Its parts consist of the fundus and its bulbous extremity which may protrude beyond the liver edge and touch the anterior abdominal wall the body is the largest part of the bladder and tapers into the neck which is much narrower Between the neck and the continuation of the gallbladder into the cystic duct is a small enlargement, the ampulla The cystic duct usually has a serpentine (coiled) appearance owing to the spiral valves of Heister on the mucosal surface The mucosa of the neck secretes an abundant supply of mucus the mucosa of the fundus does not. The gallbladder usually touches the duodenum and frequently the hepatic flexure of the transverse colon The cystic duct may lie upon

the hepatic duct for some distance before entering it. The cystic artery a branch of the hepatic usually lies to the left and cephalad to the cystic duct. However many variations of this pattern are seen and are considered normal The common bile duct is a continuation of the common hepatic duct after it has received the cystic duct It passes in the hepatoduodenal ligament (lesser omentum) in a relatively constant relationship to two other important structures The hepatic artery lies immediately medial and to its left the portal vein lies dorsal to it about halfway between it and the hepatic artery The common duct passes downward and has three distinct portions The first is in the hepatoduodenal ligament, the second is a retroduodenal portion and the third is a transpancreatic portion where it traverses the substance of the pancreas before entering the descending duodenum obliquely through the wall of the latter The supraduodenal or intrahepatoduodenal ligament portion is usually the longest and is the part most frequently inspected for pathologic changes Dorsal to the hepatoduodenal ligament is the communication between the greater peritoneal cavity and the lesser omental bursa, through a passageway known as the *epiploic foramen of Winslow* The retroduodenal portion of the common duct may be inspected by reflecting the duodenum medially after incision of the peritoneum overlying the right border of the duodenum The transpancreatic portion of the duct is in intimate relationship with the gastroduodenal artery and one of its branches the superior pancreaticoduodenal branch The duct may enter the duodenum through the wall of this structure in conjunction with the main duct of the pancreas The two enter the duodenum on a little hillock of mucosa, the papilla of Vater This entrance the ampulla is guarded by a circumferential muscle the sphincter of Oddi The two ducts the common and the main pancreatic may enter the duodenum without communicating with each other

The arterial blood supply of the extrahepatic biliary passages is derived from the celiac axis through the common hepatic trunk The hepatic artery gives rise to its two main branches the hepatic propria and the gastroduodenal The hepatic propria gives rise to the right gastric artery on the pyloric side of the lesser curvature and then divides into the two main branches of the hepatic artery which penetrate the substance of the liver parenchyma. The cystic artery most commonly arises from the right hepatic branch and arborizes over the cystic duct and the gallbladder The gastroduodenal branch lies very close to the common duct and supplies it its terminal branches are the right gastroepiploic and the superior pancreaticoduodenal The abundant lymphatics of the gallbladder cystic duct hepatic and common bile ducts drain not only to the lymph nodes in the hilus of the liver but also directly into the parenchyma of the liver

Surgical Physiology

The extrahepatic biliary ducts and the gallbladder are a hydrodynamic system for the conduction concentration and ejection of bile into the alimentary tract This is made possible by (1) the secretory pressure of the liver cells (2) the contraction pressure within the lumen of the gallbladder and (3) the normal resting pressure in the common bile duct, together with (4) the reciprocal innervation of the sphincter of Oddi Liver bile is manufactured as a dilute solution (about 3 per cent) of bile salts and acids pigment, cholesterol and lecithin in the amount of about 1 000 ml per day The principal function of bile is the emulsification of fats in the intestine through the action of its salts The liver cell secretory pressure is about 30 cm H_2O The normal resting pressure within the duct system is 8 to 10 cm H_2O *The gallbladder is caused to contract* by the absorption from the duodenum of a hormone (cholecystokinin) elaborated in its mucosa as a response to the presence of fat its pressure under such contraction rises to 25 cm H_2O This increase in its pres-

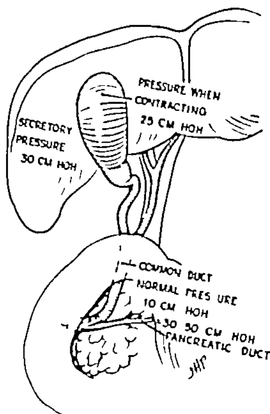


FIG 50 Hydrodynamics of the liver pancreas, and extrahepatic biliary systems. Note that the secretory pressure of the liver exceeds that of the common duct and even of the gallbladder when it is contracting The pancreatic duct pressure normally exceeds that of the extrahepatic biliary system thus ordinarily preventing reflux of bile into the pancreas

sure empties its concentrated bile which together with the reciprocal relaxation of the sphincter of Oddi admits the concentrated bile into the duodenum (Fig 50) The gallbladder stores the liver bile during the intervals between digestion In order to accomplish this despite its limited capacity it concentrates this material to as much as 10 per cent of its original volume The fatty acids and salts in bile hold cholesterol in solution

Bile pigment is a degradation product of hemoglobin released from erythrocyte break down The pigment, or *heme radical* is attached to a specific globin The degradation of the erythrocyte is carried out in two stages First, the reticuloendothelial system breaks the heme ring reducing it to a bilirubin and globin complex Second in the liver the polygonal cells separate the bilirubin pigment from the protein molecule The bilirubin pigment is converted to urobilinogen through the action of bacteria in the intestinal tract A small amount of urobilinogen is reabsorbed through the intestinal mucosa and part of this is excreted in the urine and part is returned to the liver Bile salts (taurocholates and glycocholates) are also excreted in the bile and are reabsorbed via the intestinal mucosa and returned to the liver The normal color of the stool is due to the degradation products of bilirubin (the stercobilins or urobilins)

CHOLECYSTITIS AND CHOLELITHIASIS

Etiology

Cholecystitis by definition is an inflammatory process of the gallbladder However the role of infection is often obscure and secondary Basically cholecystic disease is a metabolic disturbance The exact mechanism of the disturbance of lipoid metabolism is unknown The initial stage appears to be a chronic alteration in the gallbladder mucosa which is of metabolic origin Evidences of inflammation primarily chronic, but occasionally acute or subacute supervene at later stages Normally cholesterol is held in solution by the bile salts When the mucosa has undergone the changes mentioned, there is apparently a selective absorption of bile salts with the water leaving cholesterol in a ratio higher than can be kept in solution This favors crystallization of the cholesterol A high concentration of cholesterol crystals in the gallbladder may then result in the formation of *cholesterosis* of the mucosa, a deposition of cholesterol beads over its entire surface If a nidus of cellular debris is present, the precipitation of cholesterol about it represents the beginning of stone formation These calculi may be in the form of concentrated sludge or gravel or may be very hard Bilirubin and salts, particularly calcium, are added to the calculus forma

tion after the cholesterol crystallizes out of solution Cholecystic disease is more common in the obese, following pregnancies and after rapid fluctuations in weight. Faceted calcium bilirubinate stones are characteristic of congenital hemolytic anemia, although they occur in patients without this hematologic abnormality

Pathology

Once calculous cholecystic disease is initiated, it may take any one of several forms If the stones lie free within the lumen of the bladder and no obstruction of the cystic duct occurs the inflammatory component of this disease may long be delayed Colic may be experienced if a stone becomes engaged within a cystic duct or within the neck of the gallbladder these structures being too small to allow its passage The colic will be terminated when the stone returns to float freely within the gallbladder or is passed If the patient is operated upon because of colic at this stage or because of x ray evidence of biliary calculi without symptoms, a gall bladder whose wall is thin, unaffected by inflammatory disease and free of pericholecystic adhesions is found

If chronic obstruction by calculus occurs, colic is usually not experienced Since the body of the gallbladder does not secrete mucus, the fundus becomes filled with a concentrated solution of calcium and cholesterol known as *milk of calcium* When the obstruction by stone is below the neck, an entirely different picture may be found The neck of the gallbladder does secrete mucus Therefore obstruction below the neck may result in the filling of the neck and fundus with concentrated bile and a large amount of mucus. This is sometimes known as *white bile* but the term should be reserved for a colorless fluid in the common duct occurring when there is suppression of bile formation in the liver Under such conditions the gallbladder may become markedly distended by ever increasing amounts of mucus which continues to be poured out The bladder may reach several times its normal distended size this is also referred to as *hydrops of the gallbladder*

When stones become impacted in their passage through the cystic duct, the latter undergoes changes similar to those found in the appendix when its lumen is obstructed by a fecalith The blood supply of the cystic duct and of the bladder may or may not be compromised by the internal pressure, and an inflammatory exudate is rapidly formed as the inflammation of the entire excretory biliary system supervenes rapidly When the abdomen is explored in this stage the gallbladder is found to be markedly thickened edematous, and lacking its normal luster The great omentum and the adjacent viscera (duodenum and stomach and hepatic flexure of the colon) may be found adherent to the inflamed bulbous gallbladder by both recent and subacute adhesions nature's way of walling off this intraabdominal inflammatory process

Occasionally when the obstructive phase of biliary calculus disease compromises the blood supply to the gallbladder rapidly especially in the elderly patient, the infection may become purulent. Under such conditions the gallbladder is a sac of pus this is known as *empyema of the gallbladder* Should the defensive mechanisms normally operative in the coelomic cavity not succeed in bringing extra sources of blood to the empyematous bladder its wall will become *gangrenous* Operative intervention at this time will reveal the gangrene of the gall bladder This stage is rarely seen except in the aged and debilitated patient.

Diagnosis

Diagnosis of cholecystic disease may be made in any of the three following phases (1) chronic cholecystitis with or without stones (2) biliary colic (3) acute cholecystitis with or without empyema. The patient with chronic cholecystitis presents a fairly typical history which may include indigestion over the recent past particularly including postcibal distress and flatulence and some degree of intolerance to fatty foods.

The very first awareness of cholecystic disease however may be the occurrence of colic a severe lancinating pain of crescendo-decrescendo type in the right upper quadrant and referred toward the right scapula This is due either to the passage or attempted passage of a stone through the cystic duct or to duct obstruction and increased tension within the gallbladder or bile passages Frequently there will be a history of chronic cholecystic disease with upper abdominal postcibal distress but no previous history of acute colic or febrile illness Acute cholecystitis is ushered in suddenly with severe epigastric and right upper quadrant pain followed rapidly by nausea and vomiting in a few hours to a day fever and leukocytosis occur

Physical examination reveals marked upper abdominal tenderness with striking muscle guarding over the gallbladder region Palpation of the gallbladder or the gallbladder area on deep inspiration aggravates the pain Fever and leukocytosis are usually present with a polymorph shift to the left scout film of the abdomen may or may not reveal the calculi but will usually give evidence of an associated adynamic ileus *Cholecystography* the visualization of the gallbladder by ingested dye material is contraindicated at this time because the gallbladder will not become filled if the cystic duct is inflamed and swollen or obstructed by calculi Additionally the associated hepatic inflammation in the presence of an acute infectious process renders it unwise to force the liver cells to perform extra work to excrete the dye material The cholecystograms are best postponed until the febrile reaction has subsided

Diseases of all the supramesocolic viscera enter into the differential diagnosis of chronic cholecystitis Extrahepatic biliary colic must be differentiated from renal colic, pancreatitis and myocardial infarction Many other causes of an acute surgical abdomen must be included in the differential diagnosis of acute cholecystitis These include (1) penetrating or perforating peptic ulcer (2) a high lying acute appendicitis (3) pancreatitis and (4) intestinal obstruction of several varieties

Treatment

The treatment of cholecystitis (and it is almost always associated with calculus) is almost always *cholecystectomy* Proper timing of the operation depends on many factors Most physicians and surgeons alike prefer to allow the acute inflammatory process to subside aided by antibiotics, decompression of the upper intestinal tract and parenteral support. The gallbladder is then removed as an elective procedure during a quiescent state usually 6 weeks after the acute episode Some surgeons prefer to perform cholecystectomy during the acute phase believing as they do that the logic to be employed is the same as in the case of acute appendicitis It is difficult to argue with this logic, provided the cholecystectomy is performed within the first 24 hr after onset of the acute illness This is because in the subacute stage the inflammatory exudate in and about the extrahepatic biliary ducts and the edema of the cystic duct and gallbladder wall are so great that technical catastrophes due to poor identification and visualization of vital structures are greatly increased It should also be pointed out that the analogy between acute appendicitis and acute cholecystitis is not a perfect one Perforation of the gallbladder is not nearly as common as perforation of the appendix, because the right upper quadrant inflammatory mass is much more easily and successfully walled off and contained locally If the patient is debilitated and aged, a lesser procedure may be expedient. This involves the operation of *cholecystostomy* merely opening the gallbladder at its fundus emptying the viscus of its contained secretions and calculi and placing a drain in its lumen to lead to the outside Cholecystectomy is much preferable to cholecystostomy provided it is deemed technically feasible and safe under the conditions found at exploration. Cholecystostomy has largely been replaced by cholecys-

tectomy for the additional reason that frequently there is reformation of stones in the remaining gallbladder

Colic of the biliary tract, as colic anywhere is treated symptomatically by drugs which act directly upon the tubular structures affected and tend to relax their musculature These include nitroglycerin and associated drugs antispasmodic drugs of the belladonna-simulating group and narcotics which also have muscle relaxant properties, such as meperidine Morphine is less effective in colic since it produces contraction of sphincter muscles in the case of the biliary tract this may further raise the pressure in the extrahepatic biliary duct system and aggravate the symptoms until the sensory reception is blunted

The Postcholecystectomy Syndrome

Postcholecystectomy syndrome is the name given to the persistence after cholecystectomy of symptoms that were present before the operation It is now accepted that such persistence may be due to one of several causes. One such cause is an *error in the original diagnosis* with continuance of the disease which caused the original symptoms This is most common in the nervous patient with noncalculous gallbladder Careful diagnostic evaluation will usually obviate this source of failure Not uncommonly the postcholecystectomy syndrome is due to a disturbance in the innervation of the sphincter of Oddi, with repeated and prolonged spasm of this muscle This may be due to disease in the vicinity of the sphincter to primary disease of the pancreas, or to spasm of the sphincter mediated through emotional factors. These disturbances are collectively grouped under the name *biliary dyskinesia* Symptoms referable to the operative area may be due to overlooked *common duct stones* An occasional cause of this distressing syndrome is the presence of a stone which has been allowed to remain in a cystic duct that was not ligated close enough to its entrance into the common duct Such an overlooked stone may produce typical colic Pain in the operative area may be due to the formation of a *neuroma* from some of the autonomic nervous system twigs that are so abundant in this area The inflammatory disease which ascends the hepatic radicals may lead to *cholangitis* a febrile disease due to spread of the infection into the biliary canaliculi and along lymphatic channels The full and bursting sensation in the right upper quadrant may be due to back pressure from the common duct into the intrahepatic biliary radicals, known as *hydrohepatosis* (analogous to hydronephrosis)

CHOLEDOCHOLITHIASIS

Diagnosis

The diagnosis of *choledocholithiasis* the presence of calculi in the common bile duct, may be obvious and easy or it may be very difficult. It may be preceded by a history of repeated attacks of colic and, frequently of known biliary tract disease The pain of common duct stone may be of greater intensity and of somewhat different radiation than that of ordinary biliary colic It is almost always referred to the right scapular region or is described as a boring through and through excruciating, and agonizing pain This severe colic is due to attempted passage of a stone through the sphincter of Oddi into the duodenum Such a calculus may become lodged in the common duct and result in obstruction If the impaction persists, the liver bile is dammed back into that organ and bilirubin is absorbed into the blood stream producing jaundice This is associated as discussed subsequently in this chapter with the presence of bilirubin in the urine and in complete obstruction, with the absence of urobilinogen in the stool and urine The symptom complex of right upper quadrant colic, jaundice bilirubin in the urine absence of urobilinogen in stool and urine elevated serum bilirubin level and generalized pruritus (due to the presence of

bile salts in the skin) is the characteristic picture of choledocholithiasis This picture is frequently followed by chills and fever with leukocytosis anorexia and profound malaise The febrile episode is due to ascending cholangitis an infection which may ascend into the bile canaliculi or reach the liver by way of the lymphatics in the porta hepatis For a differential diagnosis of jaundice see the discussion in the section on Jaundice below

Treatment

Common duct obstruction must be treated by operative removal of the obstruction in order to reverse the pathologic physiology that has been productive of both local and systemic signs and symptoms The procedure is generally agreed upon choledochotomy (opening of the common duct and removal of the stone) and choledochostomy (drainage of the common bile duct to the outside) Even more than in acute cholecystitis the *timing* of the operation is important There is no emergency in operating on a patient because of a rising level of jaundice It is much more important to protect the liver against damage and support it with parenteral administration of glucose vitamin K and antibiotics When hepatic function is relatively stable and infection largely controlled operative intervention is carried out The common bile duct is explored and emptied of its calculi This may occasionally be difficult, since an impacted stone at the ampulla of Vater is occasionally difficult to recognize by palpation and when located is not infrequently very difficult to dislodge Transduodenal exploration of the ampulla of Vater with delivery of the stone may be necessary Radiographic examination of the extrahepatic biliary tract by immediate *cholangiography* on the operating table is frequently of value in visualizing stones which cannot be palpated Biliary dyskinesia of the sphincter of Oddi may be corrected at the same time preferably by transduodenal sphincterotomy (see Chapter 34)

COMPLICATIONS OF BILIARY TRACT SURGERY

One of the most serious complications in biliary surgery is stricture of the common bile duct It occurs as a result of (1) injury during cholecystectomy by laceration or ligation (2) obliterative stricture consequent upon choledochotomy in an infected field and (3) chronic cholangitis Stricture of the choledochus requires plastic reconstruction of this very important excretory duct This may be accomplished by mobilization of the retroduodenal or transpancreatic portions and anastomosis to the proximal portion in the porta hepatis When no duct can be found or mobilized a new duct may have to be created using a piece of small bowel in its stead This end is accomplished by the anastomosis of a portion of jejunum, Roux en Y fashion to a hepatic duct in the porta hepatis or to an intrahepatic duct dissected out from the liver parenchyma. Should common duct obstruction and cicatrix occur while the gallbladder is yet intact, the bladder may be anastomosed to the stomach, duodenum or jejunum for decompression of the liver

Cholangitis itself is a complication of common duct disease and common duct surgery Smoldering cholangitis over a prolonged period may lead to serious hepatocellular damage

JAUNDICE

Types and Mechanisms

Jaundice is the staining of the skin and other integumentary tissues, as well as the deeper tissues with bile pigments It is due to the retention of bilirubin in the blood stream in excessive quantities There are three main types, each with a distinctive mechanism which produces it

Hemolytic jaundice is a disorder in which there is an excessive breakdown of red blood cells with the ensuing production of

such an enormous amount of the bilirubin-protein radical that the liver cannot dissociate this complex and excrete the bilirubin rapidly enough This may be a congenital and familial condition (spherocytosis) or the result of toxins either chemical or bacterial and occasionally a manifestation of hypersplenism. The excess accumulation of bilirubin in the blood stains the tissues Bilirubin usually does not appear in the urine, because the kidney filter is impervious to the bilirubin protein complex In this disease there is no primary disturbance of the liver or bile passages This type of jaundice is also referred to as *acholuric* (meaning no bile in the urine) or *prehepatic jaundice*

Retention jaundice is due to intrahepatic disease of the liver parenchyma and is usually known as *hepatocellular jaundice* It is due to interference with function of the polygonal cells of the liver by infectious agents, hepatotoxic substances, and possibly by nutritional deficiencies In recent years the causative role of certain virus strains has been well established

Regurgitation jaundice is due to an obstruction to the outflow of bile from the liver, most commonly in the extrahepatic biliary system It is therefore also known as *extrahepatic* or *posthepatic jaundice* Clinical diseases which most commonly result in obstructive jaundice are (1) choledocholithiasis (stones in the common duct) (2) malignant tumors of or surrounding the extrahepatic biliary system these include cancers of the ducts and gallbladder of the ampulla or papilla of Vater in the duodenum and of the head of the pancreas and all metastatic lesions obstructing these pathways (3) benign stricture of the common bile duct congenital or acquired (4) obstruction by benign inflammatory disease of the head of the pancreas

In any type of chronic jaundice that has existed for considerable periods of time (as with many other chronic pathologic processes) it may not be found in pure form Thus when hepatocellular disease of the liver parenchyma has become chronic the small bile canaliculi may be obstructed by edema and stasis with resulting elements of regurgitation. Under these conditions there is a combination of primary parenchymal damage and subsequent excretory difficulties So also when the primary disease is extrahepatic biliary obstruction that has become chronic the increased pressure in the ductal system may have led to degenerative changes in the polygonal cells and have, in addition given rise to ascending infection, both of which inject an element of hepatocellular damage into what was originally a pure obstructive disease

Differential Diagnosis of the Clinical Finding

The differential diagnosis of the various types of jaundice frequently poses a difficult and extremely important problem As has been stated in the previous chapter failure to recognize and surgically relieve obstructive jaundice may be disastrous conversely laparotomy in the presence of severe hepatocellular disease may throw an added burden on the already damaged hepatic system, which may make the difference between recovery and death In most instances with careful evaluation of the history physical findings and pertinent laboratory data, the differentiation can be made without recourse to surgical exploration Occasionally it remains for exploratory celiotomy to make the differential diagnosis.

Among the important points to elicit in taking the *history* from a patient with jaundice are the following (1) previous episodes of jaundice and at what age they occurred a history of hemolytic crisis in childhood (2) evidence of extrahepatic biliary system disease as suggested by subjective manifestations and x ray evidence of biliary disease (3) earlier cholecystectomy and/or common duct surgery with the possibility of common duct stricture or residual calculi (4) blood or plasma transfusions within the past 2 to 6 months suggesting the possibility of an infectious hepatitis (5) the possible exposure to drugs known to be hepato-

toxic (6) the presence of a large and tender liver suggesting inflammatory disease of the parenchyma

Physical examination may aid in the differential diagnosis of jaundice mostly with regard to abdominal findings upon palpation A distinctly *nodular liver* suggests metastatic malignant disease A large smooth tender liver is characteristic of hepatitis The palpation of a consistently enlarged gallbladder is suggestive of malignant obstruction of the common duct (Courvoisier's law) This is so because the malignant obstruction of the common duct due to malignancy either of the common duct itself or of the pancreas is unremitting and does not allow the bile to drain from the gallbladder as may occasionally occur in calculous disease in which the gallbladder may intermittently be palpable Evidence of collateral circulation around the liver and in the abdominal wall (caput Medusae esophageal varices) and ascites suggests intrahepatic obstruction of Laennec's or virus cirrhosis types The association of chills and fever with the onset of jaundice is suggestive of cholangitis subsequent to common duct obstruction When the jaundice is progressive and unremitting, with clay-colored stool and dark urine malignant obstruction of the extrahepatic biliary system is suggested Generalized pruritus suggests obstruction since itching is almost always due to the presence of bile salts in the skin

Laboratory data which are useful in the differential diagnosis of jaundice include (1) radiologic evidence and (2) chemical data Roentgenographic evidence of calculi in the gallbladder would make one lean toward the diagnosis of extrahepatic biliary obstruction. Cholecystography is usually not helpful in the diagnosis of the cause of jaundice and may be deleterious because of the extra load the excretion of the dye imposes upon the already damaged polygonal cells Failure to visualize the gallbladder does not necessarily indicate cystic duct obstruction or disease of the gallbladder wall When jaundice is due to hepatocellular disease there may be inability of the parenchymal cells of the liver to excrete the dye Intravenous cholangiography for visualization of the common duct if positive may be very helpful Cholecystographic visualization may be helpful and safe in the presence of mild jaundice without hepatic dysfunction

The *chemical data* sought for in the laboratory are grouped together under the heading *liver function tests* or *liver profile* These attempt to measure some of the known functions of the liver both excretory and metabolic Of the *excretory tests* the most important is probably the level of the serum bilirubin This pigment, which has been dissociated from its protein fraction is normally excreted from the liver into the intestinal tract When the extrahepatic biliary tract is obstructed there is resorption, to varying degrees of this pigment into the circulatory system of the liver Normal serum bilirubin values vary between 0 1 mg per 100 ml to 1 1 mg per 100 ml The fractionation of the bilirubin into the direct and indirect (van den Bergh) is of limited value except in an uncomplicated hemolytic jaundice The direct bilirubin is a measure of pigment that has been dissociated from protein the indirect bilirubin distinguishes pigment which is still attached to the protein fragment (bilirubin globinate) and has not yet been acted upon by liver cells The *icterus index* is a colorimetric method of estimating the amount of bile pigment in the serum by comparing it with standard solutions of potassium dichromate It is less accurate than the determination of the serum bilirubin level

Alkaline phosphatase levels in the serum are helpful in the determination of the obstructive character of jaundice This enzyme is produced in bone and other tissues as well as in the liver Since it is excreted by the liver obstruction in the extrahepatic duct system produces elevation of the serum alkaline phosphatase level Lesser degrees of elevation of this enzyme in the blood are encountered in other types of liver disease although extreme degrees of retention may

be seen in severe hepatic disease The retention of a dye material Bromsulphalein (BSP) is fairly well standardized in the normal to 5 per cent or less of a standard test dose in a period of 45 min The retention of more than this amount in this time period is taken as evidence of liver cell damage

Examination of the urine and stool for evidence of disturbances in bile pigment metabolism is extremely simple and of prime importance In hemolytic jaundice (usually in childhood) bilirubin is formed in unusually large amounts urobilinogen is found markedly increased in both stool and urine There is no bilirubin in the urine because the bilirubin protein molecule does not pass the renal barrier In hepatotoxic disease there is an increase in both urobilinogen and bilirubin in the urine and urobilinogen is present in the stool In complete extrahepatic biliary duct obstruction, whether the result of stone or malignant disease there is a large amount of bilirubin in the urine and no urobilinogen in urine or stool When the common duct obstruction is intermittent or ball valve in type the urobilinogen in both the urine and stool will wax and wane with passage of the bile around the stone into the duodenum

The *metabolic functions* of the liver form the basis for still other laboratory tests One of the most important of all liver function tests is the *level of plasma proteins* particularly the albumin fraction The normal protein concentration (see Chapter 5) ranges from 6 to 8 Gm per 100 ml Of this amount normal serum albumin concentration varies from 3 to 4.5 Gm per 100 ml and normal globulin concentration from 2.5 to 4 Gm per 100 ml In parenchymal liver disease a hypoalbuminemia is characteristic This usually results in a reversal of the normal albumin/globulin ratio This reversal may also be caused by an abnormally large concentration of globulins which are manufactured largely in lymphocytes Another important protein function of the liver is the formation of *prothrombin* provided that a sufficient amount of vitamin K is absorbed from the alimentary tract *Hypoprothrombinemia* is characteristic of liver cell damage (and occasionally due to absence of bile salts in the intestinal tract) Fat metabolism in the liver is most easily assessed in the laboratory by the level of blood cholesterols In extrahepatic obstruction the total cholesterol level may rise since cholesterol is a normal component of bile However in disease of liver parenchyma there is a drop in total cholesterol levels, even if the serum bilirubin level rises. Much but not all, esterification of cholesterol occurs in the liver The normal percentage of cholesterol in the serum found as esters is about 70 per cent In general, low levels of cholesterol esters in the blood indicate liver dysfunction The relationship of the ability of liver cells to esterify cholesterol to levels of bilirubin in the blood is not well understood, but this determination is one of the earliest and most reliable indications of impaired liver cell function The several *flocculation tests* indirectly reflect liver dysfunction Actually abnormal flocculation tests merely indicate alterations in circulating serum proteins and lipid materials The cephalin-cholesterol flocculation test is positive in primary hepatocellular damage because of a qualitative alteration in the albumin fraction On a scale of 1 to 4+ readings of 2+ or more, at 48 hr are considered positive. The thymol turbidity flocculation test is positive as a result of abnormal globulins The occurrence of positive cephalin-cholesterol flocculation and thymol turbidity flocculation tests together are very strong evidence of parenchymal liver disease

From what has been stated, it is apparent that the signs of hepatocellular jaundice and that due to obstruction overlap widely and a differentiation on this basis alone may be misleading A most important consideration is the *time factor* Early obstruction of the bile tracts is not as a rule associated with impaired function Conversely a liver seriously enough diseased to produce jaun

dice will show signs of impaired function even before the jaundice becomes manifest. Therefore, a consideration of the chronology as well as the specific individual findings may point to the primary disturbance.

Surgery in the Presence of Jaundice

Surgery in the presence of jaundice is rarely an emergency. Of paramount importance when contemplating surgical intervention for therapy (or possibly diagnosis) of a patient with jaundice is the maximal possible restoration of the functional capacity of the liver. Under conditions of liver inadequacy the total body response to major surgical procedures is impaired with respect to infection, shock and general stress. The patient is best prepared preoperatively by an attempt to maintain nutrition in its every phase at the highest possible level. This includes the oral intake of a diet high in protein and carbohydrates and relatively low in fats. It is well established (see Chapter 5) that correction of depleted protein stores is far more difficult of achievement by the parenteral route than via the alimentary tract. The most effective protein restoration is accomplished parenterally by the administration of whole blood. It is wise to administer large amounts of glucose intravenously preoperatively in jaundiced patients. Vitamins, particularly synthetic K and B complex, must be added, the former to overcome the bleeding tendency in jaundice and the latter for general metabolic requirements. Antibiotics are used when indicated. Plasma protein levels and albumin/globulin ration, together with prothrombin level must be repeatedly checked and brought as close to normal as possible before operative intervention can safely be undertaken. When hepatitis is present, gamma globulin and steroids are indicated.

The role of surgery in the correction of jaundice and its deleterious effects is largely limited to the relief of extrahepatic obstruction. If stones are present in the common duct they should be removed. Strictures must be corrected by whatever means may be appropriate to permit the free flow of bile into the intestinal tract. Short-circuiting procedures may be corrective or palliative depending on the basic pathology. Drainage of bile to the outside is only a temporizing expedient and should not be considered definitive.

CANCER OF THE EXTRAHEPATIC BILIARY TRACT

Cancer of the gallbladder and of the extrahepatic ducts is usually not diagnosed preoperatively. The symptoms of these malignant diseases do not differ strikingly from calculus disease of the gallbladder and common duct. Carcinoma of the gallbladder is usually found in association with stones present in the gallbladder for a prolonged period. Eighty five per cent of all cancers of the gallbladder present this association. Cancer of the common bile duct or one of the main hepatic ducts is occasionally found upon exploration when the diagnosis of common duct obstruction by calculus has been made. Cancer of the papilla of Vater produces similar manifestations and is discussed in Chapter 35.

The treatment of cancer of the gallbladder and the extrahepatic ducts is most unsatisfactory. Direct involvement of the liver via lymphatics from the extrahepatic biliary system usually results in the finding of gross hepatic metastasis at the initial exploration. Cures are uncommon. Palliation, however, may greatly ameliorate the distress, particularly that due to pruritus, and relieve the hydrohepatosis and ascending infection. This may be accomplished by external drainage of the duct or gallbladder or preferably internal drainage of these structures to the upper intestinal tract.

SUMMARY OF PRINCIPLES

1 The extrahepatic biliary passages are a collecting, storage, and conduit system for the conduction of bile from the liver to the duodenum Their blood supply is from the branches of the hepatic artery and the system is drained by lymph nodes in the hilus of the liver and by lymph channels directly into the parenchyma

2. The hydrodynamics of the extrahepatic biliary system provide for storage of gallbladder bile until such is needed in the alimentary tract The bile is ejected from the gallbladder under stimulation of cholecystokinin, manufactured in the duodenum

3 The principal function of bile is the emulsification of fats in the intestinal tract through the action of its salts Fatty acids and salts in bile *hold cholesterol in solution The bile pigment is a degradation product of* hemoglobin

4 Basically cholecystic disease is a metabolic disturbance The exact mechanism of the disturbance in lipoid metabolism is unknown Infection may or may not supervene Calculous disease of the gallbladder begins with the crystallization of cholesterol and its deposition on the mucosa or within *the lumen of the bladder*

5 Calculi in the gallbladder may lie free may obstruct the neck of the gallbladder or the cystic duct or may pass into the common duct Obstruction of the gallbladder between the neck and the fundus results in the finding of milk of calcium in the fundus obstruction below the neck may result in the formation of a bag of white mucus (hydrops of the gall bladder)

6 Infection of the gallbladder may become purulent Empyema and gangrene of the gallbladder are usually found in the elderly and debilitated patient in whom the defensive mechanisms are inadequate

7 Diagnosis of cholecystic disease may be made in the chronic cholecystitis stage in the stage of acute colic or as acute cholecystitis with or without empyema.

8 In the differential diagnosis of acute cholecystitis are included (*a*) penetrating or perforating peptic ulcer (*b*) a high lying acute appendicitis, (*c*) pancreatitis and (*d*) intestinal obstruction In the differential diagnosis of colic renal or ureteral colic and myocardial infarction must be considered

9 Cholecystography is contraindicated during the phase of acute cholecystitis first because the edematous cystic duct may not permit passage of the dye into the gallbladder and most particularly because the excretion of the dye may impose an added burden on an infected liver

10 The treatment of choice in subsided acute cholecystitis and in cholecystic disease with calculi is cholecystectomy Cholecystectomy is usually performed during a quiescent stage Cholecystostomy may be performed as an expedient in the very ill and debilitated patient

11 Biliary colic should be treated by drugs which relax the musculature of the sphincter of Oddi such as nitroglycerin the belladonna group and narcotics which have muscle relaxant properties morphine should not be used since it produces contraction of sphincter muscles.

12 The postcholecystectomy syndrome is the name given to the persistence after cholecystectomy of symptoms that were present before the operation This may be due to (*a*) a mistaken original diagnosis (*b*) biliary dyskinesia (*c*) overlooked stones in the remnant of the cystic duct or the common duct (*d*) a residual cystic duct stump and (*e*) a neuroma of the stump Choledocholithiasis (calculi in the common duct) usually leads to colic and to signs of obstruction of the common duct including jaundice Ascending cholangitis and liver damage in prolonged cases may ensue. The treatment is common duct exploration and removal of the stones usually followed by drainage of the common duct to the outside.

13 Immediate cholangiography on the operating table is frequently of value in visualizing stones of the common duct which cannot be palpated

14 One of the most serious complications of biliary surgery is stricture of the common bile duct It may result from (*a*) injury during cholecystectomy (*b*) obliterative stricture due to choledochotomy in an infected field or (*c*) chronic cholangitis This serious complication requires reconstructive surgery

15 Jaundice is the staining of the skin and other integumentary tissues as well as the deeper tissues with bile pigments It is due to the retention of bilirubin in the blood stream in excessive quantities

16 There are three main types of jaundice hemolytic retention, and regurgitation

17 Hemolytic jaundice may be due to a congenital disorder in which there is an excessive breakdown of red blood cells with the ensuing production of enormous amounts of bilirubin protein radicals that the liver cannot dissociate and excrete It may also be of the acquired type as in some forms of hypersplenism Retention jaundice is due to hepatocellular damage of the polygonal cells by infectious or hepatotoxic agents and possibly by nutritional deficiencies In recent years the causative role of certain virus strains has been well established Regurgitation jaundice is due to an obstruction to the outflow of bile from the liver most commonly in the extrahepatic biliary system The most common causes are stones in the common duct and malignant disease in and around the extrahepatic biliary system Benign strictures of the common bile duct congenital or acquired and obstruction by pancreatitis are also etiologic

18 In long-standing obstructive disease of the common duct there may supervene an associated liver cell damage In chronic hepatocellular disease intrahepatic biliary canalicular obstruction may supervene.

19 The differential diagnosis of jaundice may usually be made taking a careful history making a complete physical examination and gathering pertinent laboratory data. Rarely exploration must be resorted to for a diagnosis

20 The history is directed toward the possible exposure to hepatotoxic drugs the presence of previous biliary disease any previous episode of jaundice and a history of aversion to fatty foods

21 Physical examination may aid in the differential diagnosis of jaundice A distinctly nodular liver suggests metastatic malignant disease A large smooth, tender liver is characteristic of hepatitis The palpation of a consistently enlarged gallbladder is suggestive of malignant obstruction of the common duct. The presence of collateral circulation around the liver in the abdominal wall and in the esophagus suggests intrahepatic obstruction of Laennec s or virus types

22 Laboratory data useful in the differential diagnosis of jaundice include radiologic evidence and chemical data Cholecystography is usually of little value in the differential diagnosis of jaundice intravenous cholangiography for visualization of the common duct may be helpful

23 The chemical data sought for in the laboratory constitute the liver function tests or liver profile These attempt to measure some of the known functions of the liver excretory and metabolic Of the excretory tests the most important are serum bilirubin alkaline phosphatase and BSP retention Examination of the urine and stool for bile pigments is simple and important In hemolytic jaundice bilirubin is found in very large amounts in the blood and urobilinogen is markedly increased in both the stool and the urine There is no bilirubin in the urine (acholuric) In hepatotoxic disease there is an increase in both urobilinogen and bilirubin in the urine, and urobilinogen is present in the stool In complete extrahepatic biliary duct obstruction there is a large amount of bilirubin in the urine and no urobilinogen in urine or stool When common duct obstruction is intermittent the amounts of urobilinogen in both urine and stool will wax and wane

24 The most important metabolic function test of the liver is the level of total plasma proteins and the albumin/globulin ratio Prothrombin level is another measure of liver function hypoprothrombinemia being characteristic of liver cell damage. Impaired fat metabolism in the liver is reflected by a lowered percentage of cholesterol esters The flocculation tests are positive in liver cell damage owing to a qualitative alteration in the protein fractions

25 Early obstruction of the bile tract is not, as a rule, associated with impaired liver function Conversely a liver seriously enough diseased to produce jaundice will show signs of impaired function even before the jaundice becomes manifest

26 Surgery in the presence of jaundice is rarely an emergency Of paramount importance when contemplating surgical intervention is the restoration toward normal of the functional capacity of the liver This is best accomplished by a high protein high-carbohydrate, low fat diet When these substances cannot be taken orally they must be administered parenterally preferably by the administration of whole blood and glucose solu

tions Vitamins particularly synthetic K to offset the bleeding tendency and vitamin B for general metabolic requirements are indicated

27 The role of surgery in the correction of jaundice and its deleterious effects is largely limited to the relief of extrahepatic obstruction This is accomplished by the removal of stones the correction of strictures and short-circuiting procedures for palliation

28 Cancer of the extrahepatic biliary tracts is frequently associated with the presence of stones over a long period of time and is rarely diagnosed preoperatively Treatment of cancer of the gallbladder and extrahepatic ducts is most unsatisfactory because of direct involvement of the liver early in the course of the disease via lymphatics from the extrahepatic biliary system directly into the liver paranchyma

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33

Diseases of the Liver (Including Hepatic Ascites and Extrahepatic Portal Hypertension)

I **Basic considerations**
- *A* Surgical anatomy
- *B* Surgical physiology

II. **Parenchymal diseases**
- *A* Hepatitis
- *B* Cirrhosis (1) etiologic and pathologic types (2) mechanism of ascites (3) treatment of ascites

III **Portal hypertension**
- *A* Etiology and pathology
- *B* Treatment of portal hypertension and its complications (1) emergent (2) definitive

IV **Infections and abscesses**
- *A* Amebic infections
- *B* Pyogenic abscess
- *C* Other infections
- *D* Infrahepatic abscess
- *E* Subphrenic abscess

V **Injuries**

VI **Tumors**
- *A* Primary
- *B* Secondary

BASIC CONSIDERATIONS

Surgical Anatomy

The liver is the second largest organ of the body weighing between 1 200 and 1 800 Gm in the adult, and is smaller in size only than the total of the integument It is a derivative of the foregut and is composed of four lobes—a large right, a smaller left, and two much smaller the caudate and quadrate Its capsule (Glisson s) is a fibrous sheath with a peritoneal investment The lobes are made up of many lobules, each of which is a functional unit which forms part of the total architecture Through the center of each runs a branch of the portal vein between the lobules course the hepatic triads, each consisting of a branch of the hepatic artery the hepatic vein and a bile duct. The lobular parenchyma is composed of cords of polygonal cells, which are the functioning elements of the viscus The sinusoids are lined by a sheath of reticuloendothelial cells (Kupffer)

The liver is in intimate relationship with many viscera Its superior surface is shaped

to accommodate the diaphragm The falciform ligament separates the right and left lobes and attaches the liver to the anterior abdominal wall Within the confines of this ligament lies the round ligament of the liver the remains of the embryonic umbilical vein On its inferior surface is to be found the gallbladder and other extrahepatic biliary passages discussed in Chapter 32 The liver is in intimate relationship with the stomach and duodenum through the gastro- and hepatoduodenal ligaments In the latter run the common bile duct the hepatic artery and the portal vein Topographically the liver is covered anteriorly largely by the right anterior thoracic cage and protrudes below it normally only in deep inspiration The vena cava traverses a part of the substance of the liver

The liver has a double blood supply It receives nutrient arterial blood from the hepatic artery a branch of the celiac axis In addition, all of the venous blood from the gastrointestinal tract is collected and brought to the liver by the portal circulation for action of the liver upon the products of digestion and absorption The hepatic artery carries 25 per cent of the blood volume to the liver but carries with it 75 per cent of the total oxygen supply to this organ All the blood leaves the liver via the hepatic veins, which join the vena cava before the latter traverses the diaphragm

Surgical Physiology

The liver is an indispensable organ of many known functions and many unknown ones Among the important known functions of this veritable chemical factory are the following (1) Carbohydrate metabolism revolves through the formation and storage of glycogen from glucose and other ingested sugars and in the reverse process, the mobilization of glycogen and its reconversion to glucose (2) Protein metabolism (catabolism and anabolism) is a continuous process in which some proteins may be stored in the liver others become deaminized with the formation of urea, and yet others are conjugated to form serum proteins (3) Bile is formed It is requisite in fat metabolism for the emulsification and absorption of intestinal lipids (4) Both coagulation and anticoagulation of the blood are affected by products of the liver the former by the formation of fibrinogen and prothrombin and the latter by the formation of heparin (5) The erythrocyte maturation factor is stored in the liver in its absence pernicious anemia occurs (6) Vitamin formation utilization and storage are a part of liver physiology particularly vitamins A and K and the B group (7) Detoxification of various noxious substances that have been absorbed from the alimentary tract occurs in the liver (8) Reversibility or irreversibility of shock is affected by the manufacture in the liver under conditions of hypoxia of a vasodepressor substance probably ferritin

PARENCHYMAL DISEASES

Hepatitis

Hepatitis is a diffuse viral infection of the liver It is increasing rapidly in incidence and severity and is becoming more frequent as the cause of hepatic cirrhosis The widespread dissemination followed the exposure of military personnel during World War II with subsequent transmission to civilian populations Two fairly distinct forms are recognized the one transmitted by homologous serum transfusion, the other of respiratory or enteric origin The former constitutes an increasingly frequent complication of blood or plasma transfusions because of the increasing numbers of carriers who cannot be detected as such by any means as yet available The incubation period in this type is thought to be longer than in the enterogenous form and usually ranges from 2 to 6 months It is also the more virulent type of infection The so-called *endemic form* has a shorter incubation period and is only relatively less severe

Pathologically viral hepatitis is a diffuse inflammation with necrosis of the polygonal

cells Both acute and chronic cellular responses are present The disruption of the hepatic parenchyma may result in jaundice which at times is severe enough to induce complete acholia of the intestinal content. If the patient survives the active phase progressive fibrosis and irregular hepatic regeneration may lead to cirrhosis and ultimately to hepatic insufficiency of a form indistinguishable from the familiar Laennec s atrophic cirrhosis Hepatitis with jaundice may at times be almost indistinguishable from extrahepatic obstructive jaundice This poses a serious problem because surgery which is essential for one may be deleterious and even fatal in the presence of the other The methodology for differential diagnosis between these diseases, based on their pathologic physiology is detailed in the previous chapter

Cirrhosis

Etiologic and Pathologic Types Cirrhosis of the liver is a disease that is clinically well recognized the pathology of it is well established, but its cause is largely obscure It is characterized by a clinical picture of a firm liver which is more frequently large than small a progressive state of malnutrition and perhaps abdominal ascites Pathologically it is recognizable as a liver which shows grossly a nubby roughened, *or hobnail surface a mottled gray and yellowish color* and histologically a marked increase in fibrous tissue parenchymal destruction and irregular areas of regeneration Sometimes referred to as *alcoholic cirrhosis* it is more commonly known after its original describer as *Laennec s cirrhosis* It is very doubtful that the specific effect of alcohol upon the liver is productive of this disease but rather there are poorly understood nutritional deficiencies that may be responsible

In recent years as is stated above another etiologic type of cirrhosis has been recognized and well documented As a part of certain systemic virus diseases there is the associated hepatitis described above, which may be followed by months to several years by the findings of an enlarged liver and abdominal ascites. Liver biopsy discloses a marked increase in fibrous tissue within this organ but, in association a picture distinctly different from Laennec s cirrhosis. There is a lymphocytic infiltration in areas of gross necrosis This picture has given substance to the description of *postnecrotic cirrhosis* of viral cause *Biliary cirrhosis* is the result of prolonged biliary tract obstruction plus the element of ascending infection The resultant inflammatory reaction around the bile ducts results in fibrosis and eventuates in jaundice and hepatomegaly The disease is usually terminated with a severe cholangitis Hanot s biliary cirrhosis originally believed to be an entity in young persons, is not recognized as such by most pathologists.

Mechanism of Ascites. Ascites is the presence of a plasmalike liquid in the peritoneal cavity It has three main causes (1) general body disturbances including particularly *cardiac decompensation* and *nephrosis* (2) *carcinomatosis peritonei* the studding of the peritoneum with malignant lesions which is probably partly inflammatory in nature and partly due to the obstruction of peritoneal lymphatics which prevents the absorption of peritoneal fluid (3) *intrahepatic disease cirrhosis which blocks the hepatic vein outflow* of blood from the liver It was incorrectly assumed for many years that portal vein occlusion whether intra or extra hepatic, caused ascites This is now known not to be true In no case does extra hepatic portal obstruction cause ascites (Fig 51) Acute obstruction of the portal vein or its tributaries usually results in hemorrhagic infarction of a portion of the alimentary tract (venous apoplexy) Obstruction of the portal system gradually over a long period of time results in the development of collateral channels, discussed later in this chapter It has been conclusively shown both experimentally and by the beau-

tiful injection-corrosion work of Madden that obstruction of the hepatic vein outflow from the liver to the vena cava is necessary for the development of ascites Peritoneoscopy (transabdominal endoscopic visualization of the peritoneal cavity) has revealed that the source of the ascites in hepatic vein obstruction is the weeping of serum through Glisson's capsule of the liver The occurrence of ascites in hepatic cirrhosis is readily explained by the fact that the hepatic vein pressure is lower than portal vein pressure and fibrous obstruction due to intrahepatic disease obliterates the hepatic

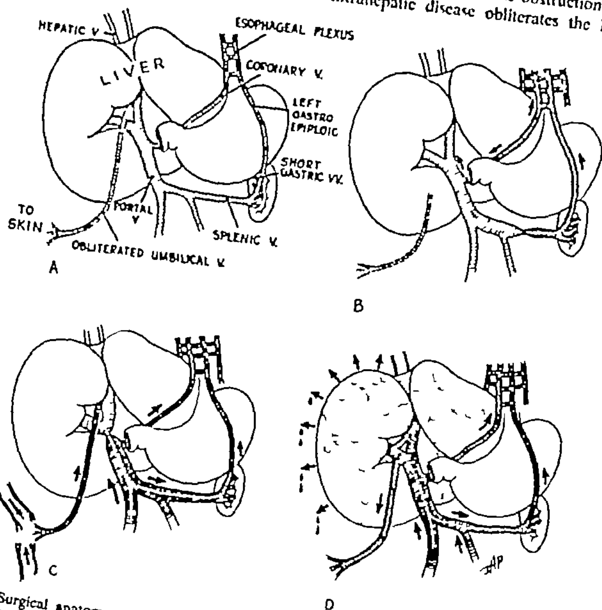


FIG. 51 Surgical anatomy and physiology of portal hypertension and of ascites. *A* Normal liver hepatic and portal circulation. *B* Partial portal system hypertension due to splenic vein thrombosis or occlusion Collateral circulation between the short gastric veins and the left gastroepiploic vein draining into the left gastric and esophageal veins, producing varices in the latter group This is the only type in which splenectomy is indicated and curative. Normal liver and hepatic veins no ascites *C* Extrahepatic portal vein obstruction producing collaterals in the esophageal and abdominal wall and hemorrhoidal anastomoses between portal and caval systems. Normal liver and hepatic veins no ascites. *D* Intrahepatic disease (cirrhosis) with intrahepatic compression of hepatic veins (note how small the hepatic veins, outside the liver have become) Blood comes into the liver but cannot get out as rapidly In this disease in addition to the resultant portal hypertension (back pressure) serum "sweats" through the surface of the liver into the peritoneal cavity This is ascites

vessels first Blood, then comes to the liver from two sources (hepatic artery and portal vein) and cannot be discharged from the liver into the vena cava rapidly enough, and ascites develops in this manner

The ascitic fluid in liver disease contains a considerable concentration of protein (about 4 per cent) This protein is lost to the body economy whether the fluid is removed or not and represents an additional nutritional deficiency

Treatment of Ascites The treatment of hepatic cirrhosis is not surgical nor is the management very effective However certain of the complications are amenable to surgical alleviation In severe ascites the peritoneal cavity may become so tense with accumulated fluid that the diaphragms are elevated and respiration compromised additionally the gastrointestinal symptoms including anorexia vomiting and the development of hemorrhoids may be aggravated by the pressure within the peritoneal cavity The removal of ascitic fluid from the coelomic cavity is accomplished by the process of *paracentesis* or *abdominal tap* This gives relief from the inconvenience and distress occasioned by massive fluid accumulation Numerous methods have been devised to permit reabsorption of ascitic fluid Most of these aim to increase the collateral venous pathways to the systemic circulation Thus, omentum has been brought into the subcutaneous space (Talma Morrison) buttons (Cooney) have been sutured between the peritoneal surfaces and subcutaneous tissues, and extraperitoneal structures, including the saphenous veins, have been brought into the coelomic cavity The success from any of these methods has not been striking Retransfusion of ascitic fluid into the patient's venous system for the purpose of conserving its protein content frequently results in reactions which are partly anaphylactic and partly foreign body In some way not yet understood the protein in the ascitic fluid is denatured and when returned to the circulating blood is more deleterious than beneficial

PORTAL HYPERTENSION

Etiology and Pathology

Portal hypertension as the term implies, is an increased venous tension in the portal bed due to obstruction to its flow into the caval circulation Such obstruction may take place within the substance of the liver as in cirrhosis, or may be in any portion of the portal vein prior to its entrance into the liver Secondary to increased venous tension is the development of elaborate collateral communications in all areas of anastomosis between the portal and caval systems. *Intrahepatic portal obstruction* occurs, as stated above as a sequel to cirrhosis of the liver Prehepatic (infrahepatic) obstruction usually results from malignant disease primary or metastatic, and usually in the porta hepatis The other etiologic factor is portal vein thrombophlebitis (pylephlebitis) and this not infrequently follows neonatal umbilical infections with cavernomatous transformation of the portal vein into a net work of interlacing small and inadequate venous channels. *Posthepatic (suprahepatic) obstruction* may occur as in Chiari's disease associated with stricture of the hepatic veins as they exit from the liver

There is normally a system of collateral vascular communications between the portal and caval systems, which becomes enormously widened to a maximal degree when the pressure in the portal system is elevated These channels include (1) the anastomosis between the inferior mesenteric (portal) and the hemorrhoidal veins (hypogastric and caval) (2) the esophageal veins (azygos and caval) with the gastric veins (portal) (3) the left renal vessels (caval) and the left colon veins (portal) and (4) the veins of the round and falciform ligaments (portal) with the superior and inferior epigastric veins of the abdominal wall (caval) Dilatation of the collateral beds in the anterior abdominal wall (caput Medusae) are of little consequence The development of hemorrhoids and their complications may be very bothersome Most

serious, however are the esophageal varices which develop as a result of dilatation of the veins draining the stomach through the esophageal veins into the azygous systems These varices are thin walled and tortuous and lie immediately under the mucosa of the esophagus and the stomach This position renders them vulnerable to erosion and results in serious and frequently fatal upper gastrointestinal hemorrhage This catastrophe is the second most frequent cause of massive upper gastrointestinal bleeding (see Chapter 31)

Treatment of Portal Hypertension and Its Complications

Emergent The management of severe bleeding from eroded esophageal or gastric varices is often most urgent and must be given immediate prior consideration This includes the temporary control of bleeding and the correction of hemorrhagic shock as has been discussed in previous chapters Control of hemorrhage is most effectively achieved by intraluminal compression through the use of a pneumatic balloon apparatus (Sengstaken) This often tides the patient over the acute emergency and permits rehabilitation and subsequent organized and deliberate definitive attack Occasionally these measures are inadequate and more heroic treatment is required This may take the form of transthoracic transesophageal oversewing of the bleeding varices

Definitive Definitive management is directed toward portal decompression by the establishment of more adequate shunts between the two systems The type of operation and the degree of success depend upon the nature and site of the obstruction. In intrahepatic obstructions the portal vein is intact, and a direct portacaval fistula can be effected. Operations designed to bypass the liver (Eck s fistula) have been employed clinically during the past 10 years Some surgeons prefer the anastomosis of the portal vein or a tributary thereof to the vena cava others prefer the anastomosis of the splenic vein (tributary to the portal) to the left renal vein (tributary to the vena cava) After the initial enthusiasm for this procedure it is now well established that it accomplishes very little when the disease is intrahepatic. However the literature on this subject lacks uniformity There are even some reports where the results seem to be better with intrahepatic disease than with extrahepatic disease Although the operation may satisfactorily decompress the portal venous system it does not affect the progression of the cirrhotic process so that the salvage is relatively short lived Those patients who respond fairly well to the operation are those who respond fairly well to medical management Those patients who are too debilitated to respond to medical management do not respond favorably to this operative procedure In general it is found that when there are less than 3 Gm per 100 ml of circulating albumin and when the ascites does not respond to a low-salt diet plus the use of diuretics, the patient will not be improved by Eck s fistula

In prehepatic obstructions which are due to carcinomatous compression of the portal vein vascular shunts are of but little value However those due to benign stricture or occlusion of the portal trunk may be given a great deal of benefit and significant prolongation of life Since the portal trunk is usually obliterated in these instances, the splenic vein must usually be anastomosed with the left renal vein to effect decompression

Occasionally a thrombosis of the splenic vein at a site before its junction with the left gastric (coronary) vein may result in the development of esophageal varices, since the splenic outflow must now reach the caval system by way of the vasa brevia and their connections with the esophageal veins Under these conditions, the hypertension and resulting varices can be cured by the simple expedient of splenectomy Under no other condition should splenectomy by itself be performed for portal hypertension, because it will fail to relieve the hypertension and, additionally wastes the splenic vein for

future use as an anastomotic channel to the left renal vein.

INFECTIONS AND ABSCESSES

Amebic Infections

Amebic infections of the liver are a result *of transmission of the causative agent, Endamoeba histolytica* from the colon via the portal vein or lymphatics. Intestinal amebiasis is treated medically Abscesses within the liver substance may require surgical therapy The abscess may attain huge proportions, is usually on the superior surface of the liver and may rupture into the free peritoneal cavity into the retroperitoneal tissues, or through the diaphragm into the pleural space The diagnosis of amebic abscess of the liver is usually based upon a febrile course with chills and sweats the elevation of the right diaphragm as determined by physical examination and roentgenographic confirmation and pain in the right upper quadrant. Leukocytosis may be high, with a relative eosinophilia. Amebae may be identified from the stool by direct smear or culture Pleuropulmonary complications, including abscess or bronchopleural fistula, may occur In the latter instance the patient coughs up the typical anchovy sauce-colored sputum Treatment is surgical and is discussed in an earlier chapter

Surgical therapy of hepatic abscesses is preceded by one or more courses of specific drug administration. The hepatic abscess may be drained transperitoneally but an extrapleural approach is preferable

Pyogenic Abscess

Pyogenic abscesses of the liver are usually multiple and are a part of a pyemic picture They are usually the sequel to a suppurative thrombophlebitis of the portal vein (pylephlebitis) most commonly due to a suppurative appendicitis Other areas of the alimentary tract are sometimes responsible for such ascending infections including the colon and the extrahepatic biliary system. Burns and other disseminated purulent processes may be the source and penetrating injuries may result in pyogenic abscesses by direct implantation of bacteria by way of the hepatic artery The diagnosis is made on the usual course and symptoms which include recurrent chills, sweats and pain in the *area of the right diaphragm, and intermittent* high fever Leukocytosis is usually high, there is tenderness over the substance of the liver and the right diaphragm is elevated Treatment includes the use of antibiotics, especially if culture and sensitivities are available Surgical drainage is only occasionally feasible because of the multiplicity of abscesses throughout the substance of the liver The latter is accomplished, again, preferably not transperitoneally but through an extraperitoneal approach. When the abscesses are multiple the prognosis is poor

Other Infections

Echinococcus or hydatid, cyst of the liver is endemic in many regions of the world where contact with the animal host excreta is common The larva reaches the liver from the intestinal tract, and cysts are formed

Diagnosis is made on the gradual enlargement of the liver a systemic eosinophilia, a positive intradermal skin test, and occasionally by x-ray evidence of a cyst in the liver Treatment is complete surgical excision of the cyst if possible and the wiping of the residual cavity with a formalin solution to kill all daughter cysts Spillage of the cyst fluid may produce severe anaphylactic shock.

Infrahepatic Abscess

Infrahepatic, or subhepatic, abscess, as a result of the accumulation of infected or purulent material in the porta hepatis, may follow any abdominal surgery or trauma, particularly of the stomach or the extrahepatic biliary system The diagnosis is usually made after such an operation when a septic course is encountered and the findings are localized to the right upper quad

rant usually with tenderness and rebound tenderness, and in the absence of evidence of change in the position or function of the right diaphragm. Treatment is almost always by re-exploration of the peritoneal cavity and drainage of the abscess.

Subphrenic Abscess

Subphrenic or subdiaphragmatic abscess is an accumulation of purulent material between the liver and the diaphragm. This space is divided into right and left by the triangular (or deltoid) ligament of the liver. The subphrenic spaces are further divided into anterior and posterior partly by the transverse limb of the same triangular ligament. The cause of subphrenic abscess is essentially the same as that of subhepatic abscess. These include any intraabdominal suppurative process, most commonly suppurative appendicitis and occasionally other ruptured viscera or intrahepatic purulent disease.

The diagnosis is made, as a rule, on the occurrence of a septic course following another disease (appendicitis, ruptured viscus, or other suppurative disease intraperitoneally or intrahepatically), usually with an intermittent spiking fever, high leukocytosis, and pain in the right upper quadrant and of diaphragmatic distribution. X-ray evidence of elevation and occasionally of immobilization of the right diaphragm must be sought for and is helpful in the diagnosis (Fig. 52). Pleural exudates, particularly on the right side, not infrequently are a sequel to subphrenic abscesses. The subphrenic abscess may even perforate the diaphragm and cause an empyema in the right pleural space. Occasionally a connection is established between the bile ducts that communicate with the subphrenic abscess and the bronchi of the right lower lobe. This *bronchobiliary fistula* is manifested dramatically by the coughing up of pus and bile and by extensive pulmonary destruction, frequently requiring pulmonary resection. Additionally, however, the subphrenic abscess must be drained and its cause removed.

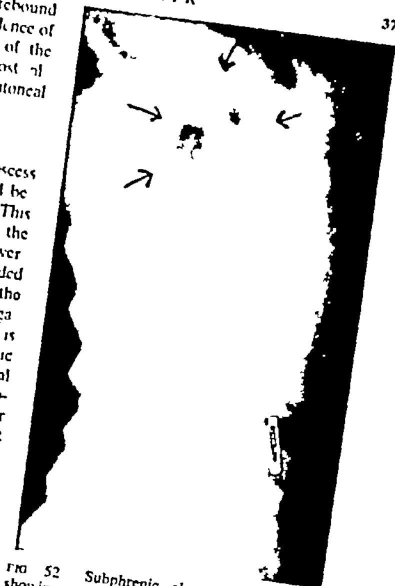


FIG. 52. Subphrenic abscess, lateral view, showing collection of fluid and gas under the right leaf of the diaphragm. Automobile accident, ruptured liver.

Treatment is appropriate use of antibiotics systemically and drainage of the abscess. The latter is accomplished preferably by an extraperitoneal approach. For the posterior superior subphrenic space this is accomplished through the bed of the resected twelfth rib and is a transdiaphragmatic retroperitoneal approach. For the anterior-superior subphrenic space a transabdominal wall–retroperitoneal approach is used. The transperitoneal and open transpleural routes are to be discouraged.

INJURIES

The liver is injured in many ways. Blunt trauma to the abdominal wall is extremely

common in military automobile industrial and athletic accidents Penetration and perforation of the anterior abdominal wall or the right thoracic cage also are frequently complicated by injuries to the liver The damage to this parenchymatous organ may vary from a small contusion to a major rupture of the viscus Between these ex*tremes there may be subcapsular hemor*rhages or intrinsic bleeding (intrahepatic apoplexy) All lacerations of the liver result in the spillage of bile as well as hemorrhage Pressure within the blood vessels of the liver is low and minor lacerations usually are rapidly sealed off by natural processes An extensive laceration may lead to rapid and irreversible shock unless whole blood is replaced very rapidly and exploration carried out promptly Biliperitoneum the presence of bile in the peritoneal cavity may be innocuous, if no infection exists and may be rapidly productive of a shock producing peritonitis if there is infection

The diagnosis of rupture of the liver is made on the history of trauma to this area of the trunk followed by pain in the area and the subsequent development of various manifestations of shock including tachycardia, hypotension pallor abdominal tenderness and rebound tenderness, indicating chemical peritonitis

Treatment of laceration of the liver depends on the severity of symptoms Minor lacerations will seal themselves major ones require prompt surgical intervention The deciding factor with regard to operative therapy and to the timing of the operative therapy is essentially the vital signs when blood is replaced, the pulse must return to normal and be maintained and there must be a subsidence of local findings in the abdomen or else exploration is urgently indicated Suture of the liver is frequently feasible at other times the laceration may be packed with hemostatic preparations such as oxidized cellulose albumin products, and thrombin If there has been much spillage of bile it is wise to drain the peritoneal cavity A severe crush of the dome of the liver as occasionally seen in steering wheel injury is best approached through the chest *and diaphragm In the postoperative period* the liver should be supported with a high-carbohydrate high protein and high-vitamin intake

TUMORS

Primary

Primary tumors of the liver are most commonly hemangiomas of congenital origin Other connective tissue benign tumors are less frequent. Primary carcinoma of the liver hepatoma, is usually engrafted upon a pre-existent cirrhosis and presumably is an expression of excessive regeneration Primary sarcomas of the liver are rare and include lymphosarcoma and hemangioendothelioma

Secondary

Secondary malignant disease of the liver is extremely common and is secondary to carcinoma anywhere in the gastrointestinal tract or pelvic viscera Such involvement of the liver is frequently palpable through the abdominal wall. Although an occasional single metastasis to the liver has been resected multiple metastatic involvement of this vital organ usually presents a hopeless picture Secondary sarcoma of the liver is most commonly a melanosarcoma originating in the eye or the skin sarcoma of bone may also metastasize to the liver The route is hematogenous the prognosis hopeless

SUMMARY OF PRINCIPLES

1 The liver is the second largest organ of the body the largest parenchymatous organ and in intimate relationship with other viscera in the right upper quadrant of the abdomen and with the diaphragm The sur

gical anatomy of the blood supply to the liver is of interest and importance the hepatic artery brings 25 per cent of the blood supply to the liver but 75 per cent of its oxygen supply the portal vein draining the gastrointestinal tract brings 75 per cent of the blood supply by volume to the liver

2 The liver is an indispensable organ of many known functions and many unknown ones The known functions of this veritable chemical factory include (*a*) carbohydrate metabolism (*b*) protein metabolism (*c*) the formation of bile (*d*) the formation of factors involved in both coagulation and anticoagulation of the blood the former by the manufacture of fibrinogen and prothrombin the latter by the formation of heparin and other substances (*e*) storage of the erythrocyte maturation factor (*f*) vitamin formation utilization and storage (*g*) detoxification of noxious substances absorbed from the alimentary tract and (*h*) the formation of ferritin a vasodepressor factor important in the irreversibility of shock under conditions of hypoxia

3 Viral hepatitis is a serious diffuse infection of the liver It has become an important cause of postnecrotic cirrhosis which clinically resembles Laennec's type although histologically it can be differentiated from it. The differential diagnosis of jaundice from causes that are surgically remediable must include viral hepatitis Cirrhosis of the liver is clinically recognizable the gross and histologic pathology are well established but its real cause is largely obscure It is usually characterized by a firm liver a progressive state of malnutrition and abdominal ascites Histologically there is a marked increase in fibrous tissue parenchymal destruction and irregular areas of regeneration

4 Ascites is the presence of a plasmalike liquid in the peritoneal cavity It has three main causes (*a*) the transudate of cardiac decompensation and nephrosis (*b*) carcinomatosis peritonei and (*c*) intrahepatic disease (cirrhosis)

5 Ascites is due to an obstruction to outflow of the blood from the liver through the hepatic veins and is therefore a result of intrahepatic disease Extrahepatic compression of the portal vein or its tributaries does not produce ascites, but it does produce an increase in the number and tortuosity of collateral circulation around the liver

6 Ascitic fluid contains a considerable concentration of protein (about 4 per cent) this protein is lost to the body economy whether the fluid is removed from the coelomic cavity or not. Its loss contributes to the nutritional deficiency of the original disease.

7 The treatment of hepatic cirrhosis is not surgical and medical management is not every effective Certain of the complications however are amenable to surgical alleviation

8 Portal hypertension is an increased venous pressure within the portal bed due to obstruction to the flow of portal vein blood through the liver and into the caval circulation Such obstruction may take place within the substance of the liver (intrahepatic) as in cirrhosis or may be prehepatic

(infrahepatic) in any portion of the portal venous system or may be supra hepatic as in Chiari s disease Esophageal varices hemorrhoids and caput Medusae of the anterior abdominal wall are expanded collateral venous pathways to return portal blood to the caval circulation

9 In intrahepatic disease shunts between the portal and caval system are of little value In prehepatic obstruction which is due to carcinomatous compression of the portal system vascular shunts are of similarly little value However those due to benign stricture or occlusion of the portal trunk may be given a great deal of benefit and significant prolongation of life If Eck s fistula cannot be made between the portal vein and the vena cava it may be made between the splenic and left renal veins.

10 The management of severe hemorrhage from eroded esophageal varices is often emergent and must be given priority The therapy includes the temporary control of bleeding by an indwelling intraluminal balloon Occasionally early transthoracic transesophageal suture of the bleeding varices may be necessary to save life Definitive management is then post poned until the patient has recovered from hemorrhagic shock

11 Splenectomy is definitive in the treatment of portal hypertension only when the blockage is in the splenic vein before its junction with the left gastric (coronary) vein

12 Amebic abscesses of the liver are usually a sequel to intestinal ame biasis Treatment of the abscess and its complications is surgical by provision for drainage of the abscess without contaminating the free peritoneal surfaces

13 Pyogenic abscesses of the liver are usually multiple and are a part of a pyemic picture They are usually sequelae of intraabdominal infectious diseases When multiple surgical drainage is only occasionally feasible under such conditions prognosis is poor

14 Echinococcus or hydatid, cyst of the liver is endemic in many re gions of the world where contact with the animal host excreta is common Diagnosis is made by the presence of a mass in the liver a systemic eosinophilia and a positive intradermal skin test treatment is total excision

15 Infrahepatic abscess is the result of accumulation of infected or puru lent material in the porta hepatis it usually follows surgery of the stomach or biliary system or infectious disease of the alimentary tract Treatment is drainage

16 Subphrenic abscess is an accumulation of purulent material between the liver and the diaphragm There is a pre-existent intraabdominal suppurative process most commonly suppurative appendicitis or rupture of some other viscus Diagnosis is made on the basis of systemic signs of an infection plus the elevation and immobilization of the right diaphragm Treatment is drainage through an extraperitoneal approach

17 The liver is injured in many ways Small lacerations are usually sealed without operative intervention Gross rupture of the liver may result

in hemorrhagic shock and biliperitoneum Under such conditions the liver must be sutured and the peritoneal cavity drained

18 Primary tumors of the liver are not common Mesenchymal tumors may be either benign or malignant the latter include lymphosarcomas and hemangio ndotheliomas Primary carcinoma of the liver (hepatoma) is usually engrafted upon a pre-existent cirrhosis and presumably is an expression of excessive regeneration

19 Secondary carcinoma of the liver is extremely common and is secondary to carcinoma anywhere in the gastrointestinal tract or the pelvic viscera Secondary sarcoma of the liver is most commonly a melanosarcoma, metastasizing by the hematogenous route

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34

The Pancreas

I. **Surgical anatomy**
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- *B* Endocrine function
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V **Cysts**
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VII **Wounds**

SURGICAL ANATOMY

The pancreas lies retroperitoneally in the epigastrium and the hypochondrium, at the level of the first and second lumbar vertebrae It is a gland which is similar to the salivary glands grossly histologically and functionally The surgical anatomy of this organ and its interrelationships with other supramesocolic viscera are as complex as those of any viscus in the body It is covered anteriorly by a sheath of peritoneum the posterior division of the lesser omental bursa Its posterior surface lies upon the prevertebral fascia It is nominally divided into a head neck, body and tail The *head* lying to the right is snugly imbedded in the concave curvature of the duodenum and touches the pylorus and the first three divisions of the duodenum Posteriorly the head lies upon the vena cava and the aorta The common bile duct traverses a portion of the head of the pancreas on its way to its oblique entrance into the descending portion of the duodenum The main pancreatic duct (Wirsung s) subserving its ex

ocrine function opens into the duodenum usually at the papilla of Vater most frequently conjointly with the common bile duct It may however enter the duodenum separately from the choledochus In either event an accessory drainage of the pancreas usually is discharged into the duodenum at a separate point (accessory duct of Santorini) Branches of the two ducts anastomose with one another within the substance of the gland The *neck* of the pancreas is a short, somewhat narrower division the posterior surface of which is in touch with the inferior border of the gastrohepatic ligament, which encloses the common bile duct portal vein and hepatic artery Dorsal to this segment the superior mesenteric and splenic veins unite to become the portal vein At the inferior border of the neck the superior mesenteric vessels emerge from behind the pancreas to cross the duodenum anteriorly The *body* of the pancreas humps over the anterior surface of the vertebral body and is the division most readily seen from the lesser omental bursa. Dorsal to the body lie the splenic and left renal vessels and the ligament of Treitz and the transverse mesocolon begin in part at the inferior surface of the body The *tail* usually reaches into the splenic hilus and is just above or adjacent to the splenic flexure of the left colon

The arterial blood supply of the head is mainly from the superior and inferior pancreaticoduodenal branches of the gastroduodenal and superior mesenteric arteries respectively The body and tail receive an abundant blood supply from the splenic artery Venous drainage from the pancreas is essentially the splenic vein plus one direct communication from the gland directly to the portal vein The lymphatic drainage of the pancreas is rich Nodes drain this organ into the hilus of the spleen all around the head along the distribution of the pancreaticoduodenal vessels, and into the preaortic nodes at the origin of the superior mesenteric and celiac arteries

The deep location of the pancreas makes it difficult of palpation for clinical differen[tial] diagnosis Its intimate anatomic relatio[n]ships with other supramesocolic visce[ra] make the diagnosis of disease of this orga[n] most difficult It is not surprising therefore that such diagnosis is often made by exclusion and not infrequently by exploratory operation For such exploration access is most easily had to the largest part of the gland through the gastrocolic ligament with entry into the lesser omental bursa The head may be inspected directly by palpation with two or three fingers in the epiploic foramen with the thumb placed anteriorly over and beyond the duodenum The tip of the tail may occasionally be inspected by exposure of the splenic hilus Examination of the inferior border of the pancreas may be made through an avascular area of the transverse mesocolon if the gastrocolic ligament is obliterated by severe inflammatory disease This approach, however is not as safe nor as adequate as can be made through the gastrocolic ligament

SURGICAL PHYSIOLOGY

Exocrine Function

The largest part of the pancreas is a gland of *external secretion* histologically a salivary type of acinous gland, producing a secretion which is active in the breakdown of all three main food stuffs carbohydrates, fats, and proteins. The pancreas produces about 1 liter of juice daily It is murky colorless, and alkaline The fraction which is concerned with the splitting of carbohydrates, *amylase* requires no activation in the intestinal tract. The ferment which splits fats, *lipase* is enhanced in its action by the presence of bile in the small intestine The potent proteolytic ferment of the pancreas *trypsinogen* is activated to *trypsin* in the small intestine by *enterokinase* elaborated by the duodenal mucosa

The secretion of these enzymes by the pancreas is under a dual control one hormonal the other nervous. The hormone

secretin is elaborated in the duodenal mucosa in response to the presence of acid chyme in the duodenum Secretin is a true hormone being absorbed into the blood stream and stimulating the acinar tissue of the pancreas The pancreatic juice so elaborated is relatively low in volume and high in enzyme content Pancreatic secretion is further under the influence of the parasympathetic system through the vagus nerve Direct or reflex stimulation of the vagi produces a pancreatic secretion which is rather large in volume but much lower in enzyme content.

Endocrine Function

The pancreas is an important gland of internal secretion, forming the hormone *insulin* in the beta cells of the islets of Langerhans This product is concerned with the metabolism of carbohydrates The beta cells are stimulated to an increased production of their specific product by the systemic level of blood sugar There are some interrelationships with other glands of internal secretion, particularly the pituitary and the adrenals, and some relationships with the liver as yet incompletely understood.

The Question of Indispensability

Whether the pancreas is an organ indispensable to life and health has not been completely answered at the present time although some light has recently been shed upon this question As with other parenchymatous organs experimental ligation of the main pancreatic ducts usually results in atrophy of the cells of external secretion leaving a fibrotic organ with normally functioning islets of Langerhans Total pancreatectomy in the human being has been accomplished it results in a diabetic state which is less severe than might be expected Total pancreatectomy in humans with preexisting diabetes has even ameliorated the disease Although some digestive disturbances follow the removal of the exocrine function as well as the mild diabetes removal of the gland is apparently compatible with life To a great extent, both the exocrine and endocrine functions can be compensated for by clinical management.

Deficient function of the "salivary" portion of the pancreas results in several types of digestive disturbances *Steatorrhea* the occurrence of frothy bulky fatty stools, is due to the presence of an excess of undigested fat *Creatorrhea* an excess of undigested protein in the stool, is the result of the absence of tryptic activity It gives rise to upper intestinal symptoms such as nausea vomiting anorexia, and pyrosis, these being the result of disturbed bowel function. Weight loss is common because of the failure of absorption of food in the gastrointestinal tract

In recent years it has been appreciated that *intractable and recurrent peptic disease* may be associated with a tumor of the non beta cells of the islets of Langerhans Resection of such tumors has effected amelioration of the ulcer disease Search for these pancreatic lesions during operation for treatment of peptic ulcer is warranted

CONGENITAL ANOMALIES

Annular Pancreas

Annular pancreas the circumferential growth of the pancreas around the duodenum is discussed in Chapter 23

Heterotopia

Pancreatic heterotopia refers to the presence of pancreatic tissue in an aberrant position Common sites of occurrence are in and around the duodenum and pylorus in and at the base of the mesentery of the jejunum in Meckel's diverticulum and more rarely in teratomas anywhere in the body

Ectopic pancreatic tissue may become clinically significant when (1) it grows large enough to simulate the presence of a tumor (2) an islet cell adenoma occurs in the heterotopic site producing hypoglycemia,

and (3) pancreatitis occurs with resulting hemorrhage

Mucoviscidosis

Mucoviscidosis is a disease of congenital origin in which there are changes in all epithelia which secrete mucus or mucuslike substances The pancreas is affected as well as the respiratory tract This disease is discussed in Chapter 23

PANCREATITIS

Pancreatitis is an inflammatory disease of the pancreas of varied causes running a whole gamut of pathologic stages with concurrent clinical pictures ranging from the acute abdomen with serious systemic disturbances to disabling and agonizing chronic syndromes

Acute

Etiology The pathogenesis of acute pancreatitis has been very widely studied Apparently many factors may be involved but an important consideration is the extravasation of activated digestive enzymes from the pancreatic duct system into the parenchyma. This process may be initiated in any of several ways The reflux of bile into the pancreatic ducts permits the activation of pancreatic enzymes by the bile salts This is made possible when a common channel exists between the main duct of the pancreas and the common bile duct before the two enter the duodenum on the papilla of Vater Obstruction of the papilla at the sphincter of Oddi, by a biliary stone edema fibrosis, or spasm in the presence of continuing pancreatic secretion is probably a more common cause of rupture of fine intrapancreatic ducts and ensuing tissue digestion Anatomic studies reveal that such a common channel exists in 20 to 40 per cent of all human beings. It is questionable whether the normal resting pancreas can be thus chemically traumatized Some evidence exists that, in order for the bile to activate the pancreatic ferments, it must be injected under considerable pressure and the pancreas must be simultaneously stimulated to activity This is usually produced by the ingestion of more than a moderate amount of alcohol together with a large meal This probably produces an excess of secretin in the duodenum The pancreas may also be stimulated to activity by the presence of an acute inflammatory process near it or upon its surface This situation obtains when a peptic ulcer of the duodenum or stomach perforates or penetrates into the head of the pancreas exciting the latter to a severe inflammatory response Mechanical trauma has also been known to excite the pancreas thus and to produce a marked inflammation of that organ In all probability such trauma is associated with rupture of either the main ducts or some of their radicles with exposure of pancreatic tissue to its own digestive enzymes

Acute pancreatitis is encountered clinically in two fairly distinct situations Association of this condition with biliary tract disease has long been recognized This is more common in females and occurs in middle and later years of life The second etiologic factor is alcoholism which may be active in either sex at any age but appears more commonly in males and in the younger age group Pancreatitis has also been laid at the door of hematogenous infection this is less common.

Pathology and Types The gross and microscopic pathologic findings in acute pancreatitis may differ greatly from case to case, from time to time in the same case and, in a given pancreas from one part of the organ to another The pathogenetic sequence appears to proceed from *edema* to *hemorrhage* to *necrosis* The latter stage represents *autodigestion* of the pancreatic substance including parenchyma, ducts and blood vessels by activated ferments of the pancreas itself The main agent in the digestive process is the proteolytic enzyme trypsin which has been converted to its active stage from the inactive trypsinogen of the resting gland by bile salts or tissue juices

Edema of the pancreas, in part or of the entire substance is an early stage recovery from this disease frequently depends on halting the process at this point Correction of the initiating cause may allow subsidence of the edema, with no progression to hemorrhage or necrosis

Hemorrhage of and into the pancreas sets off a cycle of events which is difficult to halt. The gland appears red to purple and the parenchyma is suffused with blood This stage proceeds to necrosis which may be spotty in character or which may involve the entire gland Liquefaction of the pancreas is rapid and its extremely active enzymes are spilled into the lesser peritoneal sac and, ultimately into the free peritoneal cavity The activated trypsin digests all protein in its pathway including the walls of blood vessels Activated lipase results in necrosis of fat. Plaques of fat necrosis are found throughout the coelomic cavity in the form of widely dispersed nodules, which are soapy to the touch, implanted on all peritoneal surfaces. These are in fact, saponified fats The pancreas itself manifests the autodigestive process with hemorrhage due to gross defects in vessel walls and degenerative changes of the parenchyma. The lesser omental bursa, and indeed the greater peritoneal cavity may be full of extravasated blood The entire process is one of autolysis, or autodigestion

Diagnosis The diagnosis of acute pancreatitis is frequently not made in the early stage of edema, since the symptomatology is mild and vague and may subside without sequelae At the other extreme in the stage of acute hemorrhagic necrosis of the pancreas the symptoms are dramatic, and the differential diagnosis is that of an acute abdomen (including perforated peptic ulcer, strangulated obstruction or mesenteric vascular occlusion) and myocardial infarction

Symptoms presented are usually an acute onset in a middle aged person with a history of biliary tract disease frequently following immoderate alcohol consumption and/or a large meal heavy in fat content Severe diffuse abdominal pain is experienced followed by nausea and vomiting Distention and extreme soreness of the abdomen follow rapidly The pain is usually characterized as through and through from the right hypogastrium to the right scapular area and is unremitting The patient can find no position which is comfortable to him He does not remain frozen as with the pain of angina, but rather thrashes about The usual dosage of narcotics gives no relief because the ordinary dosages of these drugs may increase the spasm of the sphincter of Oddi and thus aggravate the increased pressure within the duct system The entire abdomen is found to be tender on physical examination although the tenderness is usually maximal in the epigastrium and hypochondrium Muscle guarding is usually prominent in the upper abdomen. The abdomen rapidly becomes silent. There is one finding in acute hemorrhagic pancreatitis which is characteristic. This is the finding of shock which is out of all proportion to all other findings. The blood pressure is low the pulse rapid the skin ashen gray and moist. It is this finding which makes necessary the differential diagnosis from acute myocardial infarction At first no mass is palpable in the abdomen Fever and leukocytosis develop rapidly The signs and symptoms of blood in the peritoneal cavity are occasionally observed, including the bluish discoloration in and around the umbilicus and in the flanks Hemoconcentration can be confirmed by laboratory procedures The *serum amylase concentration* is elevated in a few hours after the onset of the attack concentration of this enzyme usually recedes and becomes normal within 48 hr Urinary amylase is elevated higher and longer than serum amylase *Serum lipase* is similarly elevated *Urinary lipase* is elevated and remains so for about a week Within a few days to a week after the onset of pancreatitis there is evidence of disturbance of the islet cells and hyperglycemia with glycosuria may

ensue Although ileus may be generalized frequently a segmental ileus of a loop or two of small bowel adjacent to the pancreatic inflammation may be visualized on an early x ray scout film Pleural effusion in the left base may occasionally be seen Within a few days after the onset of hemorrhagic necrosis of the pancreas there is deposition of ionic calcium in the saponified fats, with a reduction in serum calcium This reduction of serum calcium must be recognized and corrected without delay

Treatment The correction of shock by restoration of normal circulating blood volume preferably with blood and electrolyte solutions is imperative The stomach must be kept empty by an indwelling tube with constant gastric suction This is the most important method of keeping the pancreas at rest by preventing the formation of secretin in the duodenum An adjunctive method of keeping the pancreas at rest is the use of anticholinergic drugs by the parenteral route Relief of pain is urgent and is best accomplished by the use of drugs which have a combined narcotic and spasmolytic effect (meperidine plus nitroglycerin) rather than by the use of morphine which is productive of spasm of the sphincter of Oddi and thus may aggravate the situation Paravertebral sympathetic (splanchnic) block is used by some surgeons others prefer continuous epidural block Each occasionally is of great value Control of infection is afforded by the parenteral use of antibiotics The detection and treatment of hyperglycemia and hypocalcemia will avoid complications to an already serious affair

Operative intervention in pancreatitis is not recommended It is only occasionally employed when its differential diagnosis from conditions amenable to surgical correction cannot be made in the presence of known biliary tract disease when it is felt that decompression of the common bile duct may relieve the pressure in the pancreatic ducts and thus prevent spread of the hemorrhagic process When this is done a drain is left in the region of the pancreas

Complications and/or Sequelae The complications of acute pancreatitis are (1) pancreatic calculi (discussed in the next section) (2) pancreatic cysts (discussed in a subsequent section) (3) pancreatic fistulae (discussed subsequently) (4) diabetes and (5) hypocalcemia

Chronic Relapsing

Chronic relapsing pancreatitis is a disease characterized by recurrent attacks which simulate acute pancreatitis and in which excruciating upper abdominal pain boring in character and described as going through and through from front to back is the main feature of the picture

Etiology Chronic relapsing pancreatitis is most frequently the sequel to an episode of acute pancreatitis usually with a history of preceding extrahepatic biliary tract disease Once the process becomes established the dietary indiscretions and alcoholic intake are no longer necessary for the precipitation of recurrent attacks

Pathology The disease is characterized by multiple obstructions of the ductal system usually with calculi although occasionally with fibrocystic changes With repeated attacks diffuse fibrosis and calcification of the gland occurs

Diagnosis. A history of extrahepatic biliary tract disease and/or a previous attack of acute pancreatitis is usually elicited. Recurrent attacks are precipitated by the use of alcohol and the ingestion of heavy meals frequently however the attacks become frequent with no such indiscretions Diarrhea is associated with the recurrences in many cases Transient jaundice may occur with an attack. During the acute phase the upper abdomen may be tender and rigid just as in acute pancreatitis Not infrequently a cyst of the pancreas can be palpated Roentgen-ray examination of the abdomen by scout film may reveal pancreatolithiasis. The inflammatory expansion of the head of

the pancreas may be reflected on upper gastrointestinal examination in the widening of the duodenal loop After many attacks the serum amylase does not become elevated with each subsequent episode Changes in the exocrine function of the pancreas are more easily demonstrated *Creatorrhea* and *steatorrhea* can frequently be demonstrated and are accompanied by weight loss

Treatment. Treatment of each recurrent attack of relapsing pancreatitis differs in no way from the original acute pancreatitis It will be found that narcotics become less and less helpful in relieving the exquisite pain Indeed opiate addiction frequently supervenes Anticholinergics, antispasmodics and sympathetic block are all useful Associated and predisposing biliary tract disease must be removed by operative measures. *Sphincterotomy* the cutting of the sphincter of Oddi is frequently helpful in providing more adequate drainage from the main ductal system of the pancreas and in eliminating the cause of pancreatic pain This is best accomplished by a transduodenal approach Any pancreatic cysts that are found should be excised and drained to the intestinal tract or to the outside Associated predisposing peptic disease of the duodenum should be treated by operative methods Occasionally relief is obtained by subtotal pancreatectomy (allowing the head to remain) and embedding of the distal end of the cut surface of the pancreas into a loop of jejunum More recently the gland has been split open widely, opening its entire ductal system and the entire raw surface of the pancreas with its open ducts has been anastomosed into a loop of jejunum for adequate drainage The latter more extensive procedure seems logical and gives promise of a higher percentage of cures. Total pancreatectomy is a last resort.

CYSTS

Etiology and Types: True Cysts (Adenomas) and Pseudocysts

Cysts of the pancreas may be true cysts or pseudocysts A *true cyst* is lined by an epithelial membrane and may be congenital (rare) or neoplastic. The latter are most commonly cystadenomas of the acinar tissue. *Pseudocysts* are not lined by a true epithelium and are the result of inflammatory disease (pancreatitis) or trauma

Pathology

The typical inflammatory pseudocyst is multilocular with many thin walls and a great deal of inflammation The cystic fluid represents pancreatic secretions and liquid cellular debris which follows an attack of pancreatitis The fluid may be serous but is frequently sanguinous as well

The neoplastic cyst may be either single or multilocular with little evidence of inflammatory process surrounding them The inner lining shows evidence of papillary growth, which histologically may show evidence of malignant change.

Diagnosis

The diagnosis of cyst of the pancreas, although occasionally easy is most frequently difficult to make The outstanding symptoms are usually pain and the pressure of a mass in the epigastrium The presence of recently acquired diabetes is occasionally helpful Gastrointestinal roentgenography will usually reveal the distortion or displacement of a part of the upper intestinal tract by a spherical or globular mass the colon may similarly be displaced *Intravenous pyelography* likewise may reveal the displacement of the left kidney Differential diagnosis usually must be made between pancreatic cyst on the one hand and renal tumors, mesenteric cysts and omental cysts on the other

Treatment

Ideally excision is the treatment of choice. More often than not however the cyst wall and its inflammatory process have so thoroughly involved the adjacent viscera including stomach duodenum spleen colon the retroperitoneal space and left kidney that attempt at excision is unwise It is much more conservative to drain the cyst either to

the outside thus creating an *external pancreatic fistula* or into a loop of upper jejunum thus creating an *internal pancreatic fistula* When an external pancreatic fistula is created the cyst may diminish rapidly in size and obliteration may be obtained by packing with an agent that produces granulation such as iodoform gauze If this end is not attained a second operation may reveal that the cyst which is now much smaller can be excised with a greater margin of safety A successful internal pancreatojejunal fistula usually requires no further treatment

TUMORS

Carcinoma

Adenocarcinoma is the common malignant tumor of the pancreas It may occur anywhere in the gland and is common in the head It is more common in the male and constitutes 1 to 2 per cent of all carcinomas found at autopsy

Diagnosis When carcinoma of the head of the pancreas involves the common duct and obstructs it early the diagnosis is made rather easily When the common bile duct is not obstructed the diagnosis may be obscure and made very late This is because the symptoms are those of diseases of many supramesocolic organs and not specific to the pancreas Additionally the pancreas is difficult to palpate because of its deep position behind the abdominal viscera in the retroperitoneal space Midepigastric pain radiating through to the back is characteristic, especially when progressive in character The pain is usually made worse after eating and in the supine position Anorexia and change in bowel habits are frequent Jaundice occurs in most cases of carcinoma of the head of the pancreas and in about three quarters of all cases of cancer of the pancreas. Anorexia, fatigue and cachexia with weight loss are late symptoms Migrating phlebitis, unattributable to local causes may be found at various stages in the disease possibly related to some chemical change in the blood-coagulating mechanism A diminished antithrombin titer may be demonstrated Unremitting jaundice together with a palpable gallbladder which never recedes (Courvoisier's law) is characteristic of carcinoma of the head of the pancreas and/or the papilla of Vater This is probably due to invasion of both the wall and the lumen of the common duct by tumor A palpable abdominal mass other than the liver and gallbladder is not frequently found Laboratory evidence of extrahepatic biliary obstruction is found in the form of an elevation of serum bilirubin and alkaline phosphatase Creatorrhea and steatorrhea are determined on examination of the stool Hyperglycemia and glycosuria may occur as the disease progresses Upper gastrointestinal roentgenography may reveal a widened duodenal loop and indeed a compression of the duodenum It is difficult clinically to differentiate the symptomatology of carcinoma of the head of the pancreas from that of the papilla of Vater In the latter occasionally ulceration of the lesion permits remission of the jaundice and serum bilirubin level Carcinoma of the papilla of Vater is really a carcinoma of duodenal mucosa The symptomatology produced by this lesion simulates that of carcinoma of the head of the pancreas in that obstruction to the outflow of bile from the common duct with ensuing jaundice occurs In tumor of the papilla of Vater the growing lesion may occasionally be visualized on x ray examination of the duodenum Additionally the stool usually contains occult blood, owing to the sloughing character of the fungating growth

Pathology The site of origin may be in ductal or acinar tissue Although adenocarcinomas or carcinoma simplex are the rule a rare epidermoid cell carcinoma is also described Invasion through the wall of the duodenum through the prevertebral fascia and into the vertebral bodies, and along the nodes of the aorta is common Lymphatic spread to the porta hepatis is the rule hematogenous metastases are frequently seen.

Treatment Ideally the treatment of cancer of the pancreas, as elsewhere is resection of all tumor bearing tissue Because of the most intricate surgical anatomy of the pancreas this is rarely feasible An occasional small tumor discovered early because of its proximity to the common bile duct, is discovered and removed and a cure effected Far more often however the extensiveness of the malignant lesion in the pancreas itself as well as in the adjacent area precludes any attempt at definitive therapy When the head of the pancreas is resected the distal end of the common duct is of necessity removed with it and a portion of the duodenum as well This combined operation pancreaticoduodenectomy is a very extensive procedure requiring much dissection and several anastomoses for reconstitution of the extrahepatic biliary system and the upper gastrointestinal tract The initial mortality is high and cures are few Probably nowhere else in the body is the dictum "large tumor—small operation small tumor—large operation " so true Pancreaticoduodenectomy is more logically applicable to carcinoma of the papilla of Vater than to carcinomas of the head of the pancreas This is because in the pancreatic lesion there usually is extension to surrounding and adjacent viscera and along the prevertebral fascia by the time the disease is first manifested by obstruction of the common duct Opposed to this, carcinoma of the papilla of Vater may be detected at a time when its removal by pancreaticoduodenectomy and the reconstruction of the gastrointestinal tract and its derivatives, offers a greater hope of survival This indeed has been found to be true in the experience of all surgeons.

Adenoma of Islet Cells

Adenomas of the beta cells of the islets of Langerhans may be found in any part of the pancreas or in any heterotopic pancreatic tissue The cause is obscure About one tenth of islet cell tumors are malignant Only those adenomas which are functioning in character come to clinical attention

The diagnosis of adenoma of the islets of Langerhans is made on a very characteristic symptomatology, in which there is fainting profuse diaphoresis tachycardia, nausea and convulsions These attacks are relieved rapidly by the intravenous or oral administration of sugar A blood sugar level determined before the therapeutic use of glucose will show a low concentration usually 25 to 60 mg per 100 ml. The symptoms described are in every way comparable to *hypoglycemia* produced in the laboratory by excessive administration of insulin

Treatment is the very careful exploration of the pancreas, with removal of all nodules and their microscopic examination Most of these tumors are found in the body and tail of the pancreas however they may be anywhere and a thorough search should be made

WOUNDS

There are two main types of injuries to the pancreas, operative and from external violence Injury to the pancreas is not infrequently sustained in many operations on upper abdominal viscera. These include those of the biliary tract, the stomach and duodenum the spleen and left kidney and more recently following extensive vascular replacement. External violence usually is in the form of blunt trauma to the abdomen and is seen not infrequently in industrial and automotive accidents Penetrating injuries, as with missiles, require little elaboration The danger of all injuries to the pancreas of course is the production of an acute hemorrhagic pancreatitis with *autodigestion* of the gland and all the sequelae described earlier in this chapter The symptoms of pancreatic trauma of any cause are those of an acute pancreatitis Whenever upper abdominal tenderness and rigidity is experienced under such conditions and the picture of shock appears to be out of all proportion to other symptoms, the diagnosis of traumatic pancreatitis must be entertained The treatment and complications are those of

acute pancreatitis of any cause Lesser degrees of trauma may produce no obvious pancreatitis but may result in the finding of pseudocysts at a later time

SUMMARY OF PRINCIPLES

1 The pancreas resembles salivary glands grossly, histologically and functionally The surgical anatomy of this organ is extremely complex It is contiguous with and borders a wide variety of structures it is affected by them and affects them These structures include the vertebral bodies and prevertebral fascia the aorta and its celiac and superior mesenteric branches the vena cava and the portal vein circulation the duodenum and stomach the lesser omental bursa and the gastrohepatic and hepatoduodenal ligaments the transverse colon and its mesocolon the extrahepatic biliary system and the spleen The pancreas is normally difficult of palpation through the anterior abdominal wall

2 The pancreas is really two glands one of external secretion the other of internal secretion The salivary portion of the pancreas produces enzymes which are active in the breakdown of all three main foodstuffs carbohydrates proteins, and fats. The hormone of the pancreas manufactured by the beta cells of the islets of Langerhans is insulin it is active in carbohydrate metabolism

3 The pancreas is not indispensable for life To a great extent, both the exocrine and endocrine functions can be compensated for by clinical management

4 Deficient function of the salivary portion of the pancreas results in several types of digestive disturbances including steatorrhea and creatorrhea in addition to upper intestinal symptoms Deficient function of the endocrine factor of the pancreas results in hyperglycemia and glycosuria.

5 Tumors of the nonbeta cells of the islets appear to have ulcerogenic properties

6 The congenital anomalies of annular pancreas heterotopia, and mucoviscidosis are discussed in detail in other chapters.

7 Pancreatitis is an inflammatory disease of the pancreas, of varied origin running a whole gamut of pathologic stages with concurrent clinical pictures ranging from the acute abdomen with serious systemic disturbances to disabling and agonizing chronic syndromes

8 The cause of acute pancreatitis may be (*a*) activation of trypsinogen to trypsin by the presence of bile in the pancreatic duct system this may be aided and abetted by the common channel between the Wirsung s duct and the common bile duct (*b*) the presence of an acute inflammatory process near or upon the surface of the pancreas this situation obtains in the presence of perforating or penetrating peptic disease (*c*) mechanical trauma to the pancreas (*d*) hematogenous infection this is less common

9 Clinically pancreatitis is seen most commonly when there is a history of pre-existent extrahepatic biliary disease in a patient whose pancreas

has been stimulated to excessive secretion by immoderate intake of alcohol and a heavy meal

10 The pathology of acute pancreatitis may vary from localized or generalized edema to hemorrhage and finally to necrosis The ultimate destruction of the gland is by autodigestion Autolysis consumes the parenchyma the blood vessels and adjacent tissue Activated lipase results in fat necrosis The split fats are saponified and united with calcium resulting in plaques of hard fat necrosis distributed throughout the peritoneal cavity

11 In acute pancreatitis shock may be out of all proportion to all other findings The pain is through and through hypogastrium to back, and is unremitting and excruciating Fever leukocytosis and signs of blood in the peritoneal cavity follow rapidly The serum amylase concentration is *elevated in a few hours and returns to normal within 48 hrs the urinary* amylase is elevated to a greater degree and for a longer period of time. Serum lipase is also elevated, and urinary lipase is elevated to a greater extent and remains so for about a week Hyperglycemia glycosuria, and *ileus may accompany the disease Hypocalcemia is common when fat necrosis* is extensive

12 The treatment of acute pancreatitis includes the restoration of normal circulating blood volume maintenance of an empty stomach to prevent the formation of secretin in the duodenum relief of pain by all effective methods control of infection and correction of hyperglycemia and hypocalcemia. Operative intervention is not recommended if it is undertaken and biliary tract disease is discovered decompression of the common bile duct is indicated together with drainage of the peritoneal cavity

13 The complications and sequelae of acute pancreatitis are (*a*) pancreatic calculi (*b*) pancreatic cysts (*c*) pancreatic fistulae (*d*) diabetes and (*e*) hypocalcemia

14 Chronic relapsing pancreatitis is a disease characterized by recurrent attacks which simulate acute pancreatitis *and which are usually a* sequel to an episode of acute pancreatitis and pre-existing extrahepatic biliary disease The pathology usually includes multiple obstructions of the ductal system frequently with calculi and almost always with fibrocystic changes Roentgenographic evidence of pancreatolithiasis creatorrhea and steatorrhea and ultimately weight loss are common

15 The treatment of chronic relapsing pancreatitis may vary from transduodenal sphincterotomy to partial pancreatectomy with the embedding of the distal cut end of the pancreas into the jejunum When there is evidence of diffuse fibrocystic disease of the pancreas with or without calculi the entire gland may be split open the ducts widely opened and the raw surface anastomosed into the jejunum Total pancreatectomy is a last resort

16 True cysts of the pancreas are lined by epithelium and are either *congenital (rare) or neoplastic Pseudocysts are not lined by epithelium* and are the result of inflammatory disease or trauma.

17 Pseudocysts of the pancreas are treated by excision if possible and

by marsupialization to the outside or fistulization to the jejunum if total excision is not possible

18 Cancer of the pancreas may occur anywhere in the gland but is most common in its head When it occurs in the head jaundice is usually the presenting symptom because of obstruction by tumor of the common duct Other symptoms are midepigastric pain radiating through to the back, change in bowel habits anorexia and fatigue migrating phlebitis and the presence of a distended gallbladder which never recedes Diabetes and a widening of the duodenal loop are occasionally seen

19 Only small and incidentally found cancers of the pancreas are successfully treated by excision, radical cancer surgery is extremely difficult in this area because of the intimate relationships with other viscera, and prognosis is poor

20 Wounds of the pancreas may be inflicted by external violence or by operative trauma The results are either pancreatitis or the formation of pseudocysts

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The Spleen

- I Surgical anatomy
- II. Surgical physiology
- III Indications for splenectomy
  - *A* Trauma (1) external violence (*a*) immediate hemorrhage (*b*) delayed hemorrhage (2) operative
  - *B* Mechanical (1) large size due to chronic infection (2) wandering spleen and twisted pedicle
  - *C* Diseases (1) hypersplenism (*a*) platelets—thrombocytopenic purpura (*b*) red series—splenic anemia (*c*) white series—splenic leukopenia granulocytic anemia (*d*) pancytopenia (2) congestive splenomegaly (Banti's syndrome portal obstruction venous thrombosis) (3) familial hemolytic jaundice (spherocytosis) (4) metabolic (5) tumors and aneurysms
- IV Contraindications

SURGICAL ANATOMY

The spleen is a parenchymatous organ purplish red in color weighing 150 to 200 Gm in the average adult and resembling the liver in consistency It lies deep and high in the left hypochondrium covered by the left anterior rib cage It moves with respiration but is palpable on deep inspiration only when enlarged

The fixation of the spleen varies greatly but its interrelationships with other viscera in the left upper quadrant are rather constant It lies close to the greater curvature of the stomach, to which it is attached by the gastrosplenic ligament which contains the *vasa brevia* that course between these two structures The tail of the pancreas is usually found in the hilus of the spleen Ligaments, or peritoneal folds attach the spleen to the kidney (lienorenal ligament) and to the colon (lienocolic ligament) and the spleen frequently rests partially within the phrenicocolic ligament.

The principal arterial supply of the spleen is the splenic artery a branch of the celiac axis which travels along the superior margin of the pancreas gives rise to the left gastroepiploic artery and then divides into its terminal branches near the hilus of the spleen Each of the main branches that penetrate the spleen is essentially an end artery with very little overlapping of circulation between the various divisions of the spleen The splenic vein follows the general course of the splenic artery it unites with the superior mesenteric vein to form the portal There are no lymphatics in the parenchyma of the spleen there are lymphatics in the splenic capsule which are drained to the nodes along the splenic artery

The structure of the spleen is adapted to the role which this organ plays in the physiology of the circulation The capsule com

posed of peritoneum and fibrous tissue gives off trabeculae which divide the parenchyma into compartments The trabeculae also contain smooth muscle cells which permit the spleen to contract The parenchyma is a spongy mass which is made up of "white" and "red" pulp The former is the Malpighian corpuscles, which consist largely of accumulations of lymphocytes and which are surrounded by red pulp composed of venous sinuses and pulp cords These cords are columns of reticuloendothelial cells monocytes, macrophages lymphocytes and some leukocytes

The arterial blood entering the spleen passes from the arterioles chiefly into the pulp cords, where it comes in contact with the active cellular components of the organ A portion of the inflow circulation is shunted through an alternate pathway into the splenic sinuses where it is believed the erythrocytes may be stored to be released by adrenergic stimuli when needed to supply systemic demand

Accessory spleens are found in a relatively large percentage of human beings. They vary greatly in size from 1 mm to 5 or 6 cm and possess the characteristic color and texture of the parent organ. Their most frequent sites are along the splenic vessels, in the hilus of the main spleen at the root of the mesentery of the small intestine especially near the ligament of Treitz. In conditions of splenic hyperplasia, there is general enlargement of the accessory splenic depots, and minute foci become large enough to be readily visible When splenectomy is performed for the purpose of eliminating splenic hyperfunction care must be exercised to remove the accessory organs also

SURGICAL PHYSIOLOGY

The spleen performs several important functions, probably including some as yet unknown. Nevertheless, it is not essential to life nor to a state of general well-being However the spleen may take over additional vital functions in certain diseases and so attain indispensability

In embryonic life the spleen is a major factor in red blood cell formation In adult life ordinarily only lymphocytes and monocytes are formed In certain diseases it may reassume its embryonic role in adult life and function not only in erythropoiesis but in the generation of all cellular elements The spleen acts as a reservoir of blood by storing red cells which may be mobilized by adrenergic stimuli under stressful conditions The splenic reticuloendothelial cells are active in several ways Hemoglobin is transformed into bilirubin as a part of the degradation process of old erythrocytes Reticuloendothelial cells also perform the functions of phagocytosis of particulate foreign bodies and the formation of antibodies The spleen also exerts a regulatory influence on the bone marrow probably through the mediation of a true hormone

INDICATIONS FOR SPLENECTOMY

Trauma

External Violence Rupture of the spleen is a frequent component of abdominal thoracic, and thoracoabdominal injuries whether blunt or penetrating It occurs most commonly following trauma to the left upper quadrant of the abdomen or to the left lower chest cage Laceration of the spleen by a fractured lower left rib is not uncommon The normal spleen is less susceptible to damage by trauma than is an enlarged friable organ Industrial and automotive injuries produce an ever increasing number of injuries to the spleen

This parenchymatous organ may suffer several types of injury Tear of the capsule with a fracture of the solid tissue of the spleen is probably most common Laceration of the vessels in the pedicle of the spleen is probably less common, but it is more serious and even rapidly fatal A break in the pulpal parenchyma of this structure, with subcapsular hemorrhage is not infrequent. The full

blown picture of trauma to the spleen ensues when the capsule finally is breached and the blood is spilled into the free peritoneal cavity

The diagnosis of rupture of the spleen must be considered in every case of abdominal or thoracic injury or in combined thoracoabdominal trauma as well as in relatively innocuous fractures of the lower left ribs. The symptoms and signs of rupture of the spleen are essentially (1) hemorrhagic shock and (2) chemical peritonitis produced by the presence of blood. The onset of shock may be rapid and profound if bleeding is from the splenic pedicle or if the peripheral wound in the spleen is not immediately plugged by omentum or clot. Contrariwise the picture of shock may develop slowly with a minimal tachycardia and hypotension in the early stages It may even appear that the patient is recovering from this state with a return of vital signs to normal and later a recurrence of unequivocal evidence of shock. This delayed shock may follow the initial episode by minutes hours, days or even weeks In the interim the fracture of the splenic parenchyma has been plugged either by a clot which is ultimately extruded by contraction of the spleen or by omentum which finally becomes loosened

The local abdominal findings are pain and tenderness in the left upper quadrant, accompanied by varying degrees of muscle guarding Pain referred to the left shoulder is due to irritation of the left diaphragm by blood The left diaphragm and thoracic cage may be splinted by pain The pain and tenderness may soon be followed by rebound tenderness and the signs may become diffuse over the entire abdomen This progression plus the systemic findings of shock make mandatory the consideration of the diagnosis of rupture of the spleen. In rapid bleeding there is no time for hemodilution and accordingly the blood profile is of little value in differential diagnosis Blood volume studies may aid the recognition of hemorrhage When the splenic hemorrhage is delayed by hours or days the hemodilution that occurs will reflect the blood loss in the lowering of the hematocrit and hemoglobin levels. Roentgenographic scout films of the abdomen may reveal a diffuse haziness due to the blood in the coelomic cavity Abdominal paracentesis in order to make the diagnosis is not often necessary but may occasionally be resorted to and yield the desired information

Treatment of rupture of the spleen is abdominal celiotomy as soon as the diagnosis is made and splenectomy The spleen is not an organ that is frequently amenable to suture When the spleen has been severely fragmented, every effort should be made to remove as many of the small dispersed fragments of splenic pulp as possible This is in order to avoid seeding of the peritoneum by little pieces of spleen which will take hold and grow resulting in the condition known as *splenosis* which is potentially dangerous if hypersplenism occurs at a future date

Operative Trauma to the spleen occasionally occurs during operative intervention upon other supramesocolic viscera such as resections of the stomach, pancreas and splenic flexure of the colon and operations for esophageal hiatus hernia. If the trauma is recognized, splenectomy should be done at that time

Mechanical

Large Size Due to Chronic Infection A large spleen is an appreciable hazard in an active person, in that it is susceptible to traumatic injury by minimal body contact. The large spleen of chronic malaria is probably the best example of such a situation and constitutes a legitimate indication for splenectomy The large spleen of congestive splenomegaly associated with hepatic cirrhosis may be an indication for splenectomy for the same reason It is not to be construed that the splenectomy in any way cures the hepatic disease This type of splenomegaly associated with hepatic disease if accompanied by anemia, is usually referred to as *Banti's syndrome* The large spleen of *Banti's syndrome* may require removal because of

signs of hypersplenism discussed in the next section

Wandering Spleen and Twisted Pedicle Occasionally a spleen is seen which is attached to its vascular supply by an unusually long pedicle Such a pedicle may become twisted and the volvulus of that pedicle may cause infarction and gangrene of the spleen Such a catastrophe produces an acute abdominal picture and requires splenectomy Short of twisting the long pedicle may allow the spleen to wander from its normal site to other places in the abdomen where it becomes both bothersome to the patient and a source of worry in differential diagnosis. Thus, a long pedicle to the spleen may constitute an indication for splenectomy

Diseases

Hypersplenism The term *hypersplenism* is applied to a group of clinical situations in which the spleen apparently exhibits an exaggeration of one or more of its normal functions Such hyperfunctioning may result in reduction of any one or all of the cellular elements of the blood When there is no disease of the spleen itself the bone marrow or any other systemic disease to account for this aberrant action the term *primary hypersplenism* is used. When the hyperfunctioning organ is associated with disease of the spleen itself such as congestive splenomegaly the lymphomas and leukemias, sarcoidosis, and myeloma the term *secondary hypersplenism* is appropriate

Methods of study of splenic function are (1) hematologic examination, including detailed study of the bone marrow and the peripheral blood for cell morphology and types, counts and fragility bleeding and coagulating profiles and the Coombs test (2) physical examination of the abdomen for enlargement of the spleen and (3) operative or percutaneous splenoportography the visualization of the spleen and the portal venous system by the injection of radiopaque materials directly into the spleen This procedure is not without obvious danger

In patients with hypersplenism the platelets alone may be affected resulting in *thrombocytopenia* and a hemorrhagic tendency (purpura) This is more common in childhood but it does occur in adults as well It is characterized by hemorrhage from any or all mucous or cutaneous surfaces or in soft tissues, as well as within parenchymatous organs These hemorrhages which may be insidious and progressive or sudden and massive occur spontaneously or with minimal trauma Diagnosis is aided by the laboratory findings of (1) a marked reduction in the number of circulating blood platelets, (2) prolonged bleeding time (3) prolongation of the clot retraction time and (4) an increase in megakaryocytes in bone marrow and an ill-explained increased capillary fragility The apparent disparity between the increased number of megakaryocytes and diminished number of platelets cannot be entirely explained by the increased destruction in the spleen There is additionally, an apparent suppression of release of platelets into the circulation from the megakaryocytes on the presumed basis of a splenic hormone present in excess The disease must be differentiated from secondary (symptomatic) purpuras, usually due to some drug or bacterial toxin

Thrombocytopenic purpura is a grave disease primarily because of the possibility of spontaneous cerebral hemorrhage and therefore merits immediate and often emergent therapy The steroids are frequently of temporary value but definitive treatment is splenectomy which may have to be performed as an emergency While this disease is still considered by most persons to be a part of the picture of hypersplenism immunochemical relationships between the spleen and the bone marrow are currently under investigation

Hypersplenism occasionally affects essentially the red blood cell series, resulting in *splenic anemia* presumably by excessive splenic destruction of erythrocytes The disease is characterized by anemia and splenomegaly The treatment is splenectomy

In *splenic neutropenia (agranulocytic anemia)* the spleen is grossly enlarged The bone marrow usually exhibits myeloid hyperplasia Examination of the surgical specimen histologically reveals the splenic pulp to be filled with a great quantity of granulocytes in various stages of degradation. Clinically there is a marked *susceptibility to infections* of all types Splenectomy is usually very helpful

In *idiopathic primary splenic pancytopenia* all the cellular elements are diminished The spleen is almost always large the bone marrow is markedly hyperplastic If the favorable response induced by steroids cannot be maintained splenectomy is the treatment of choice

Congestive Splenomegaly (Banti's Syndrome; Portal Obstruction, Venous Thrombosis) Congestive splenomegaly *Banti's syndrome* an enlargement of the spleen with an associated clinical anemia and leukopenia, with or without hepatic cirrhosis is now considered to be merely the effect of portal hypertension upon this organ Occasionally esophageal varices are encountered in the presence of congestive splenomegaly Suffice it to say here that the operation of splenectomy for esophageal varices and portal hypertension is curative only when the venous obstruction is in the splenic vein distal to the joining of that vein with the left gastric vein When such a situation obtains the vasa brevia are the only venous pathways carrying blood from the spleen and their communications with the left gastroepiploic vein and the phrenicoesophageal veins are the only means of venous drainage from the spleen Splenectomy then obviously removes the cause of such varices

Familial Hemolytic Jaundice (Spherocytosis) A blood dyscrasia of unusual character is congenital (familial) hemolytic jaundice also called *primary hemolytic jaundice* It is a chronic disease associated with varying levels of jaundice a large spleen and persistent anemia, spherocytic erythrocytes and the frequent association of calcium bilirubin type of gallstones Acute exacerbations of the hemolytic process are frequent Laboratory evidence of the disease includes the recognition of red blood cells which are spherocytic rather than ovoid in shape the increased fragility of the red cells and the presence of both immature erythrocytes and a high reticulocyte count in the peripheral blood. *The disease has a hereditary tendency* The apparent mechanism for the congenital hemolytic jaundice is the increased tendency of the spleen to destroy erythrocytes in less than their usual life span of 120 days An excess of the hemoglobin complex in the blood results in an abnormally high concentration of bilirubin in the blood which is responsible for the clinical picture of jaundice Remissions of crises are occasionally possible with the use of steroids. Blood transfusions should not be given during crises since the transfused cells may also be hemolyzed Splenectomy is preferably accomplished during a period of relative quiescence The jaundice of course is cured but the spherocytosis remains for life

Metabolic Splenectomy is indicated in certain metabolic diseases associated with splenomegaly Removal of the spleen must be performed early in these diseases if it is to be helpful at all Late in the progress of these diseases it may be only palliative or not effective The diseases are essentially those in which there is a faulty lipid and carbohydrate metabolism, with the packing of reticuloendothelial cells with huge amounts of these materials This accounts for the alternate name of *lipoid histiocytoses* Included are Gaucher's disease, Hand Schüller Christian disease Niemann-Pick disease and Letterer Siwe disease Splenectomy late in the progress of any of these conditions is technically difficult, since there is a marked perisplenitis, with adhesions to the abdominal wall the diaphragm and all upper abdominal viscera.

Tumors and Aneurysms. Primary tumors of the spleen are rare Most tumors of the spleen are in the lymphoma group, and evidence of one of these diseases is usually available elsewhere in the body Tuberculo-

sis of the spleen is not uncommon is never an isolated finding and requires systemic medical therapy Sarcoidosis is occasionally responsible both for a hypersplenic syndrome and for a bothersome large spleen which must be removed Abscess of the spleen is usually secondary to tuberculosis

Splenectomy is indicated when an aneurysm of the splenic artery is found It is considered safer and subject to fewer complications than vascular resection and replacement.

CONTRAINDICATIONS

The contraindications to splenectomy are mostly relative although an occasional absolute one exists Relative contraindications are essentially those in which the pathology is not clear and the results poor and equivocal. These include thalassemia (Mediterranean or Cooley's anemia) and sickle-cell anemia A clinical finding which is usually considered to be an absolute contraindication to splenectomy is the finding of *myeloid metaplasia* of the spleen In this disease the spleen resumes its fetal state in manufacturing blood elements of the myeloid series This is usually associated with a complete suppression of the myeloid forming elements of the bone marrow or with an aplastic anemia in which there is little if any bone marrow In this situation the spleen is performing a function of the bone marrow If removed, death due to infection usually supervenes rapidly

The surgeon should at all time work in close liaison with the hematologist with respect to diseases of and which affect the spleen In general it is well to remember that a good contraindication to splenectomy is the absence of a clear-cut indication

SUMMARY OF PRINCIPLES

1 The spleen is ordinarily not essential to life Only under certain circumstances when it assumes embryonic functions, does it attain indispensability

2 The spleen has a number of known functions and some as yet unknown

3 The spleen may act as a reservoir of blood by storing red blood cells which may be mobilized by adrenergic stimuli under stressful conditions The histology of the spleen is well adapted to this function

4 The reticuloendothelial cells of the spleen perform a number of its functions Included in these is the transformation of hemoglobin into bilirubin as a part of the degradation process of old erythrocytes phagocytosis of particulate matter and the formation of antibodies

5 It is probable that there is an interrelationship between the spleen and the bone marrow of immunochemical importance probably on a hormonal basis

6 Fracture of the spleen is common in abdominal thoracic, and thoracoabdominal injuries The hemorrhage may be immediate or delayed The characteristic findings are shock and abdominal tenderness

7 In the performing of splenectomy for fracture of the spleen all fragments of pulp should be removed in order to avoid splenosis at a later date

8 A large spleen such as that found in chronic malaria is a legitimate indication for splenectomy

9 The wandering spleen on a long pedicle is also an indication for

splenectomy because it may become twisted and lead to parenchymatous infarction

10 Hypersplenism is the term applied to a group of recognizable clinical findings in which the spleen apparently exhibits an exaggeration of one or more of its normal functions In hypersplenic diseases any one or all of *the blood cellular elements may be reduced in number* When the platelets are so affected, the disease entity is known as *thrombocytopenic purpura* When the red series is affected, the name applied is *splenic anemia* When the white series is affected the name *splenic neutropenia* is appropriate When all cellular elements are involved the disease is called *splenic pancytopenia*

11 Congestive splenomegaly or Banti s syndrome, is the presence of an enlarged spleen plus the clinical findings of anemia and neutropenia, with or without hepatic cirrhosis The congestion and its sequelae are probably the result of portal hypertension

12 Familial hemolytic jaundice is a congenital disease in which there is a structural alteration in the red cell, anemia increased destruction of red blood cells, and an enormously increased amount of bilirubin in the blood

13 There is a group of diseases, of metabolic nature involving essentially the histiocytes in which a large spleen is produced These include Gaucher s Hand Schüller-Christian, and Letterer Siwe disease

14 Tumors of the spleen and aneurysms of the splenic artery are not common but require splenectomy for treatment.

15 When the spleen reassumes its fetal function of manufacturing myeloid or erythroid series elements (as in aplastic anemia) splenectomy is contraindicated

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Hernia

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- II Types
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  - *A* Indirect
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- V Femoral hernia
- VI Umbilical hernia
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Hernia is the most prevalent disease for which surgical management is required. It affects both sexes, all ages, and persons in every walk of life. Since it is largely due to certain congenital predispositions, it is very unlikely that means of preventing it will be found in the foreseeable future. It is, furthermore, a mechanical defect, for which the development of effective nonoperative therapy is scarcely to be anticipated.

DEFINITION

Hernia is usually defined as the protrusion of an organ from the cavity in which it is normally contained. This definition is obviously incorrect and misleading. It misleads by focusing attention on the protrusion rather than on the defect in the wall through which the protrusion occurs, which is the actual lesion. It is incorrect in that the protruding viscus is often reduced into its normal habitat, either spontaneously or by manipulation. Yet no one would claim that the hernia had been cured when this occurs. A more inclusive but somewhat unwieldy definition is that *hernia is a defect in the wall of a cavity through which some contained structure has protruded and will protrude again if the pressure within is sufficiently high.*

TYPES

The term *hernia* is sometimes used with reference to the cranial or thoracic cavities. However, the overwhelming majority of hernias occur in the abdominal walls, and only abdominal hernias will be considered in this chapter. There are many varieties of abdominal hernia which differ sufficiently from one another to require separate discussion.

Among these varieties are *inguinal femoral umbilical ventral diaphragmatic obturator* and *perineal* The hernial aperture or parietal defect, varies in these different forms Often *the opening is an enlargement or incompetency* of a normal hiatus In others it may be congenital or traumatic, whether surgical or accidental

COMPLICATIONS

Certain complications may occur though *in different degrees of probability in various* hernia types If the contents of the hernial protrusion can be returned to the abdominal cavity the hernia is said to be *reducible* However in many instances the contents cannot be reduced for numerous reasons Such hernias are called *incarcerated* or *irreducible* Incarceration may be due to disparity *between the size of the extruded contents* and that of the orifice to adhesions of the viscera to the *sac* i.e a pouch of peritoneum which passes through the defect and contains the herniated structures or to relative immobility of the organ occupying the sac (*vide infra* sliding hernia) The most frequent organ extruded in a hernia is the small bowel Because of kinking adhesion or constriction at the neck of the sac such a loop of intestine may become obstructed, giving rise to an *incarcerated hernia with intestinal obstruction Strangulation* is the most serious of the complications affecting the contents of hernias As the name implies, the bowel is so constricted at the hernial opening or ring as to occlude its circulation This constitutes a surgical emergency since strangulation unrelieved will lead to gangrene of the involved bowel segment In every series of intestinal obstruction cases external hernia is one of the most frequent etiologic factors An appreciable mortality rate attends all intestinal obstructions and particularly strangulations Timely repair of hernias before these accidents occur is an effective means of reducing the incidence and thereby preventing deaths from intestinal obstruction

The term *sliding hernia* is applied when a portion of a partly retroperitoneal structure such as ascending or descending colon, is drawn into the sac together with its peri*toneal covering It thus comes to constitute* part of the wall of the sac The significance of this type of hernia lies in the necessity of recognizing it when it is encountered at operation and in returning the contained viscus to the abdomen without injury to it or to its circulation.

INGUINAL HERNIA

Inguinal hernia is somewhat loosely defined as a hernia in the inguinal region More specifically it is a hernia protruding through the *inguinal triangle* This refers to the triangular space bounded superiorly by the lower free margin of the internal oblique *muscle medially by the edge of the rectus* sheath, and inferiorly by the inguinal ligament (Fig 57) The significance of this geographic entity will be made clear below

Inguinal hernia comprises by far the largest number of all hernias As is seen in Table 4 it constitutes 94 per cent of hernias in the male 43 per cent of hernias in the female and 83 per cent of all hernias. Because of its relative importance the inguinal variety will be presented in greater detail

There are two varieties of inguinal hernia, the *oblique or indirect* and the *direct* Except for the fact that they both emerge through the inguinal triangle they are entirely different diseases, each with a different congenital predisposition different anatomy and different pathology Their clinical manifestations and predilections toward complications differ and they should have correspondingly different surgical management Only by considering each in its pure form can they be made entirely clear Later the combined forms can be better understood.

Indirect

Indirect inguinal hernia is best defined as herniation into the spermatic cord To completely understand this form of hernia, as

TABLE 4

RELATIVE INCIDENCE OF VARIOUS TYPES OF HERNIA

Type	Per cent total	Per cent males	Per cent, females
Inguinal	83	94 0	43
Incisional	7	2.5	26
Femoral	6	2 0	17
Umbilical	4	1.5	14
Total	100	100.0	100

well as certain other abnormalities of the groin and scrotum a precise knowledge of the descent of the testicle as it migrates *through the abdominal wall to reach the* scrotum is essential The embryonic testis *forms* within the abdomen and lies behind the parietal peritoneum As the organ en larges it *moves downward and at the third* month it lies adjacent to the abdominal inguinal ring It passes through the aponeu rosis of the transversus abdominis muscle at this level carrying with it the ductus deferens *and* spermatic vessels and a fold of peritoneum which is called the *processus vaginalis* (Fig. 53) From the investing fascia of the transversus abdominis the *forming* spermatic cord derives its innermost investment the internal spermatic fascia When the level of the internal oblique muscle is reached the descending testis passes *under* the lower free margin of this muscle and the cord derives from it a second investment, a musculofascial membrane known as the *cremasteric layer* At the subcutaneous inguinal ring, the migrating structures pass between the crura of the ring receiving as they do a third investment from the epimysium of the external oblique aponeurosis, which becomes the external spermatic fascia. Thus, *prior to birth a channel has been formed,* the inguinal canal which passes obliquely

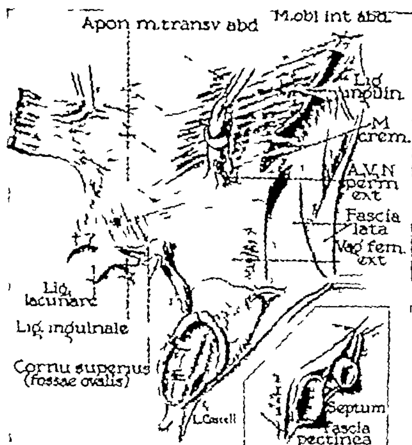


FIG. 53 Abdominal inguinal ring from the external surface. The cord structures together with a contained patent processus vaginalis pass through the aponeurosis of the transversus abdominis muscle taking with them their innermost investment, the internal spermatic fascia, which is derived from the transversalis fascia. (*From McVay and Anson Anat Rec* 76 213–231 1940.)

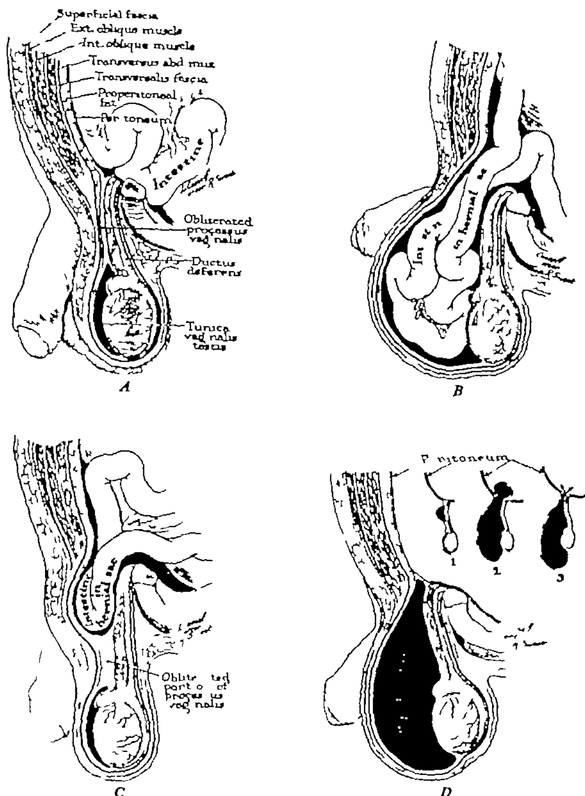


FIG. 54 *A* Complete obliteration of processus vaginalis testis *B* "Congenital" type of indirect inguinal hernia Processus vaginalis patent throughout its course. C "Acquired" type of indirect inguinal hernia with funicular process open above but obliterated below *D* Funicular process closed above and patent below producing hydrocele (1) Encysted hydrocele of cord from partial obliteration of processus vaginalis. (2) Infantile type of hydrocele (3) Communicating hydrocele (*From Anson and Maddock Callander's Surgical Anatomy W B Saunders Co* 1952.)

through the abdominal wall and which is occupied by the spermatic cord with its several investments containing the vas deferens, spermatic vessels and nerves and a peritoneal pouch which is continuous with the peritoneal cavity above and with the space between the parietal and visceral layers of the testicular tunics below The former of these layers ultimately becomes the tunica vaginalis testis and the latter, the tunica albuginea. At birth the peritoneal process normally becomes obliterated leaving only the potential space between the two testicular membranes

In the female although no gonad migrates through, a similar channel or canal forms which contains the round ligament of the uterus a corresponding peritoneal pouch, and identical investments Closure of this processus vaginalis normally occurs also at about the time of birth

In a very significant percentage of persons of both sexes closure of the processus vaginalis fails to occur The persistent patency may be total or partial and may involve various portions of the pouch (Fig 54) Patency of the proximal portion of the processus, which communicates with the peritoneal cavity is a *sine qua non* for the development of an indirect inguinal hernia. If the entire processus is patent, the basis is present for the so-called *congenital indirect hernia* that is one in which the sac is continuous with the parietal peritoneum

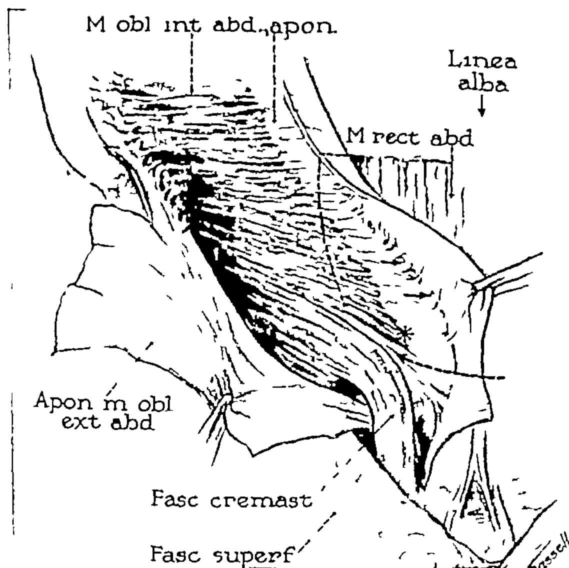


FIG. 55 Strong type of internal oblique muscle completely covering the lower abdomen No tendency toward direct hernia. Narrow inguinal triangle (*From Anson and McVay Anat Rec.*, 70 211–225 1938)

above and with the testicular membranes below If only the proximal portion remains open the resulting hernia is called *acquired indirect hernia* although it is no less congenital in its origin than is the type described above Other variations as shown in the illustration result in *congenital hydrocele multiple encysted hydroceles of the cord* or the *communicating hydrocele* of the newborn A persistent processus vaginalis in the female is known as the *canal of Nuck* and similarly predisposes to indirect hernia. It must be emphasized that the presence of a preformed sac is not synonymous with hernia. Many such sacs remain unoccupied by abdominal content throughout the life of the individual A hernia is present when first abdominal content enters the sac. Conversely no indirect inguinal hernia develops in the absence of such a sac. Strength or weakness of the abdominal musculature has no bearing on the development of this type of hernia

On the basis of what has been said, it is possible to reduce the pathology of uncomplicated indirect inguinal hernia to but two deviations from the normal—a preformed sac (the *persistent processus vaginalis*) and a moderate dilatation of the *abdominal inguinal ring* The abdominal inguinal (internal) ring is the funnel shaped hiatus in the transversus abdominis aponeurosis through which the cord passes to begin its egress through the abdominal wall Normally this aperture is just large enough to transmit the cord It is protected by the internal oblique muscle which arches over it When the muscle contracts, it descends over the ring in *the nature of a shutter* When abdominal contents enter the sac and move back and forth, there is stretching and widening of the ring The enlargement cannot take place upward because of the protection afforded by the internal oblique muscle It necessarily therefore enlarges downward, so that it extends beyond the overhanging internal oblique muscle and comes more and more to overlie the *subcutaneous (external) ring*

Direct

Direct inguinal hernia as has been stated, is an entirely different disease from the indirect variety having little in common with it, other than its topographical location It, too has a congenital predisposition but one that bears no relation to the spermatic cord or the descent of the testis The predisposition to direct hernia consists of an *absence of the fibers of the internal oblique muscle or its aponeurosis* in the lowermost portion of the abdominal wall. The muscle pattern shown in Figure 55 in which the internal oblique fibers extend down to the pubis and leave but a narrow slit between the muscle and the inguinal ligament just large enough to accommodate the spermatic cord, is never associated with a direct hernia. On the other hand a pattern such as that in Figure 56 in which the lowermost internal oblique fibers insert horizontally into the rectus sheath, leaving a large triangular area devoid of muscle support and protected only by the transversus aponeurosis, is very liable to direct herniation. This is the muscle pat

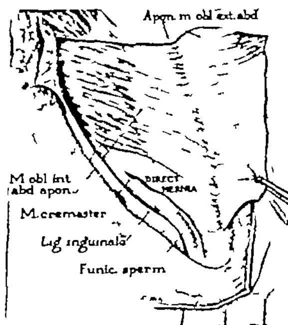
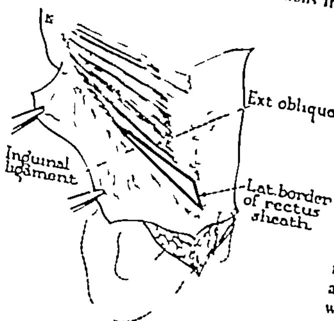


FIG. 56. Muscle pattern which predisposes to direct hernia. Lower fibers of internal oblique muscle absent leaving broad inguinal triangle (*From Anson and McVay Surg Gynec & Obst.*, 66 186–191 1938)

tern always found at operation on patients with direct inguinal hernia The inguinal triangles in these two types of abdomens differ strikingly from one another (Fig 57)

The pathology of direct hernia can thus also be reduced to but two deviations from the normal First there is the muscle pattern which makes for weakness of the abdominal wall The posterior wall of the area, made up of the unsupported transversus aponeurosis alone is insufficient to withstand the intraabdominal pressure and it bulges outward below the lowermost margin of the internal oblique Thus, the second component in the pathology is the stretching and tearing of the transversus aponeurosis



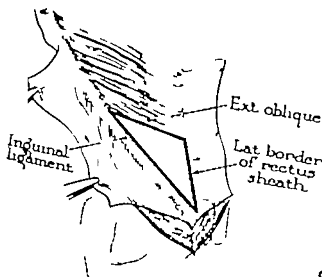


FIG 57 Inguinal triangle bounded above by lowermost fibers of internal oblique muscle rectus sheath medially and inguinal ligament laterally *Above* Type of inguinal triangle which may be present with indirect hernia but does not predispose to direct hernia *Below* Inguinal triangle always associated with and giving rise to direct hernia (*From Zimmerman S Clin North America* 32 135-153 1952.)

Both congenital predispositions to indirect and direct varieties of inguinal hernia are very common so that in a certain number of persons both will be present Such persons may have both forms of hernia simultaneously or they may have one and a predisposition to the other so that a combined repair is necessary It is also conceivable that a massive long standing indirect hernia with perhaps the added trauma of years of truss-wearing may sufficiently attenuate and destroy the musculature to render the abdominal wall as vulnerable as if a direct hernia had also been present.

Treatment

Over the centuries the operative repair of inguinal hernia has vied with retentive management by means of a truss In the light of modern achievement, there is but one acceptable therapy namely *early and adequate surgical correction* There is no place for the truss as treatment for hernia except for the very occasional instance when operation must be postponed because the physical condition of the patient does not warrant the intervention The tendency to delay operation once the diagnosis has been made is to be deplored Nothing is gained by such procrastination It involves expense and inconvenience to the patient and only defers the repair to a worse time Patients become older physical impairments supervene the hernia becomes larger and the tissues poorer and the ever present hazard of strangulation lurks in the background What might have been a simple repair of a small hernia in a

young person with an excellent promise of permanent relief becomes a difficult operation often mandatory for a voluminous hernia in an old subject who may have developed serious systemic impairment and poorly healing local structures Extremes of age mark no exception to the rule Whether infants or persons "on borrowed time" operative repair is the indicated treatment for inguinal hernia.

Many methods have been advanced for the repairs of hernia, including different technics, suture materials, and a host of prosthetic substances An enumeration or evaluation is beyond the scope of this book. However it is pertinent to point out the requisites of adequate operative procedures, in terms of the essential pathology of the two forms of inguinal hernia As has been pointed out before the essential pathology of indirect hernia is a preformed sac and a dilated internal ring Logical repair therefore would entail removing the sac and narrowing the ring to normal size Direct hernia consists in wearing through the posterior wall of the canal because of lack of muscular support. The indications therefore are to repair the transversus aponeurosis and to reinforce it with some type of plastic procedure that will give it sufficient strength to withstand permanently the abdominal pressure To the extent that both types coexist, a combination of the methods should be employed.

FEMORAL HERNIA

Femoral hernia is much less common than the inguinal variety but it carries a twentyfold propensity for incarceration or strangulation This type is much more frequently seen in females than in males However in women it occurs less often than do inguinal hernias It is characteristically a disease of adults.

Femoral hernia is a hernia through the *femoral ring into the femoral canal* which is a conical space within the *femoral sheath* This sheath is a fibrous investment for the femoral vessels, the anterior half of which is derived from the aponeurosis of the transversus abdominis and the posterior half from the pectineus fascia. The opening into the cone-shaped canal is at the level of the superior ramus of the pubic bone and is bounded by the inguinal ligament anteriorly the superior pubic ramus posteriorly the sharp crescentic edge of the lacunar (Gimbernat's) ligament medially and the femoral vein laterally It is because of the essentially rigid and unyielding character of this ring that incarceration and strangulation so frequently occur

Femoral hernia is usually easily differentiated from the inguinal variety if it is reducible If the mass is followed with the finger as it returns to the abdomen, the opening will be found just below the medial end of the inguinal ligament in inguinal hernia it lies just above the ligament If the hernia is not reducible, the differentiation may be more difficult. Other conditions which may simulate femoral hernia are varicosity of the long saphenous vein at the fossa ovale and inguinal adenopathy

Operative repair is mandatory whenever femoral hernia is found because of the great danger of strangulation. The operation may be done through a femoral incision below the inguinal fold or through an inguinal incision, which is carried through the posterior wall of the inguinal canal into the preperitoneal space above the inguinal ligament The sac is excised in either operation and the femoral ring closed

UMBILICAL HERNIA

Umbilical hernia as the name implies, is a herniation through the *umbilical ring* This ring is formed by protrusion of abdominal content through the scar left by the sloughing of the umbilical cord *following its ligation* The peritoneum is adherent to the inner surface of this scar and the skin to its outer

Umbilical hernias are encountered in three stages of life at birth (congenital type) in early childhood (infantile) and in adults *Congenital umbilical hernias* are relatively

rare and represent failure of the anterior abdominal wall to close owing to persistence of the *exocoelom*. Between the sixth and tenth week of intrauterine life the abdominal viscera grow at a more rapid rate than does the coelomic cavity and part of these structures are temporarily housed in the exocoelom, a peritoneum lined space which is continuous with the abdominal cavity and which occupies the base of the umbilical cord. If the disparity in size persists beyond the tenth week the exocoelom prevents the closure of the musculofascial structures and skin of the anterior abdominal wall.

Omphalocele

At birth in such infants, there is a parietal defect, with a protruding mass occupying

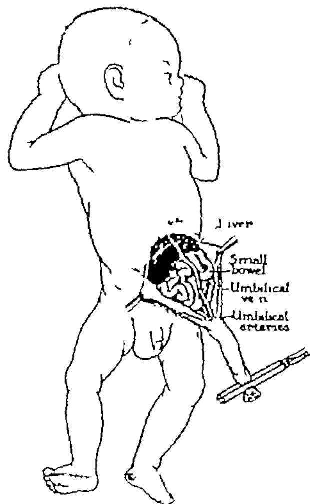


FIG. 58 Large omphalocele in newborn infant, containing a large portion of the abdominal viscera. (*From Zimmerman and Anson Anatomy and Surgery of Hernia Williams & Wilkins Co* 1953.)

the base of the umbilical cord (Fig. 58). Through the transparent membranes may be seen the visceral structures which may include virtually all of the abdominal organs. The term *omphalocele* is applied to these anomalies, which may vary in size from 4 to 15 cm in diameter. Uncorrected such defects are incompatible with life since the thin membranes are easily ruptured exposing the peritoneal contents. Immediate surgery is necessary. Small defects are easily closed by suture; large ones may be impossible to close except by a two-stage procedure. At the time of birth the skin is widely mobilized on both sides and the edges brought together by suture leaving, in fact, a large ventral hernia. The parietal closure is effected 6 to 24 months later when the abdominal cavity is large enough to accommodate all its proper contents.

Infantile Umbilical Hernias

Umbilical protrusions are extremely common in infants during the first few months of life. They usually disappear before the age of one year whether kept conscientiously strapped or ignored. A very small percentage, however, either persist or recur several years later and such hernias may attain sufficient size to require surgery.

Adult Type

Adult hernias at the umbilicus may become very large, not infrequently strangulate and occasionally stretch the overlying skin until it becomes thin, atrophic, and ultimately ulcerated. Such hernias should be operated upon. In all umbilical hernia operations the technic usually consists of imbrication, or lapping the upper edge of the defect over the lower to provide a two-layer aponeurotic closure.

VENTRAL HERNIA

Hernia through the anterolateral walls of the abdomen may be spontaneous or postoperative and may involve the midline or the lateral surfaces. *Spontaneous midline hernias* occur in the linea alba, almost always

in the upper abdomen between the xiphoid process and the umbilicus Because of their location they are frequently called *epigastric hernias* Such hernias occasionally attain considerable size but for the most part they remain small. They may be solitary or multiple and probably represent failure of fusion of the two halves of the abdominal wall Because of the sharply defined margins of the hernial rings, incarceration frequently occurs the contents consist in part or entirely of preperitoneal fatty tissue The only treatment for such hernias is operative repair

Spontaneous

Spontaneous lateral ventral hernias are much less common than those of the mid line These are often referred to in the literature as *hernias of the semilunar line* (*of Spigel*) or as *Spigelian hernias* It has been shown (Zimmerman and Anson) that these hernias are due to a characteristic pattern of the internal oblique and transverse abdominis muscles. In a not inconsiderable number of persons the fibers of these muscles are not arranged in a continuous sheet as is usually depicted in the anatomical drawings but are divided into "bands" somewhat reminiscent of the somatic bundles with defects between the successive muscle bands If the defects in the two muscle layers are superimposed one over the other a basis for herniation is provided These hernias are usually *interstitial* in that their sacs spread out beneath an intact external oblique aponeurotic layer

Incisional

Incisional hernias are due to defective healing of incisional scars in the abdominal wall They are second in frequency of all hernia forms, owing to the very large number of abdominal operations being done the most massive of all hernias fall into this group They are characteristically multilocular with extensive adhesion of intestinal loops to the lining of the sac, and, consequently prone to bowel obstruction and strangulation Operative repair too is often difficult and tedious and intestinal injury and extensive oozing from denuded peritoneal surfaces are not infrequent. For all these reasons incisional hernias carry a material mortality and therewith a challenge to the surgeon

Incisional hernia is closely linked to the problems of wound healing and wound dehiscence both of which are discussed in earlier chapters Among the etiologic factors making for poor healing are hypoproteinemia, vitamin C deficiency and allergy to absorbable suture material Postoperative factors result from excessive distention severe coughing and vomiting and uncontrolled agitation and straining From the standpoint of operative technic, there must be mentioned the selection of incision and suture material the meticulousness of the manipulation, and the care exercised in the closure Debilitated persons and particularly cancer patients are apt to display poor healing tendencies

Incisional hernias vary enormously in size and, correspondingly in problems of management Very large defects may be beyond repair unless foreign prosthetic materials such as metal or plastic meshes, are utilized Prevention is better than repair and to this end the principles of surgery are indispensible, including adequate pre and postoperative management and the observance of the best of operative technic.

SUMMARY OF PRINCIPLES

1 Hernia is a *defect* in the wall of a cavity through which some contained structure has protruded and will protrude again if the pressure within is sufficiently high

2 Abdominal hernias may be inguinal femoral umbilical ventral diaphragmatic obturator or perineal

3 If hernias cannot be reduced they are called *irreducible* or *incarcerated* These may or may not cause intestinal obstruction Strangulated hernias are incarcerated with the added factor of obstruction to the blood supply of the contained structures

4 Timely repair of hernias is an effective means of preventing strangulation with its inevitable considerable mortality

5 Inguinal hernia constitutes by far the most frequent type of hernia It may be of the indirect (oblique) or direct variety

6 Indirect hernia is a herniation into the spermatic cord or its analogue in the female It is always due to a persistent patency of the processus vaginalis

7 The pathology of uncomplicated indirect inguinal hernia consists of the patent processus vaginalis and a secondary dilatation of the abdominal inguinal ring Its repair requires removal of the sac (the processus vaginalis) and closure of the abdominal ring to a normal size.

8 Direct inguinal hernia is a weakening of the posterior wall of the inguinal canal owing to absence of the lowermost fibers of the internal oblique muscle or its aponeurosis. Repair requires closure of the defect in the aponeurosis of the transversus abdominus and adequate plastic reinforcement to prevent subsequent herniation

9 The treatment of inguinal hernia is operative repair as soon as the presence of the hernia is recognized

10 Femoral hernia is a hernia through the femoral ring and into the femoral sheath

11 Femoral hernia is much more susceptible to strangulation than is the inguinal variety and, therefore, *must* be operated upon when first discovered

12 Umbilical hernias may be congenital (omphalocele) infantile, or adult.

13 Ventral hernias may be spontaneous or postoperative (incisional) The spontaneous variety may occur in the midline (epigastric) or laterally The latter form are due to "banding" of the internal oblique and transversus abdominis muscles, with hiatuses between the muscle bands

SUGGESTED READING

ZIMMERMAN L. M AND B J ANSON *Anatomy and Surgery of Hernia* The Williams & Wilkins Company Baltimore, 1953

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